SLEEP, EATING BEHAVIOUR AND WEIGHT IN EARLY CHILDHOOD

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Declaration

I, Laura McDonald, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

Acknowledgements

To Professor Jane Wardle, thank you for giving me the opportunity to pursue my PhD within the HBRC. It was an immense honour and a privilege to have been able to work with you. Your loss is felt deep within the group, and in your passing I am acutely aware of the extent to which you prioritised the success and wellbeing of your students. Your unsurpassable work ethic, vast knowledge, and empathetic approach to your students was an inspiration. I hope that I am able to carry forward your influence into all my future work.

To my supervisors Dr Abi Fisher and Dr Clare Llewellyn, thank you for your expert guidance throughout, and for helping me to develop as an independent researcher. To Abi, with whom I have worked most closely with over the past few years, it has been an absolute pleasure. I feel incredibly lucky to have had such a genuinely kind mentor, whose unwavering support extended far beyond the academic.

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This thesis is dedicated in fond memory of Professor Jane Wardle (1950 – 2015)

Abstract

The rising prevalence of obesity poses a considerable threat to population health. Shorter nighttime sleep has emerged as a novel risk factor for overweight and obesity, and the association appears to be stronger at young ages. Experimental studies in adults suggest that increasing energy intake drives weight gain during periods of sleep curtailment. However, there have been few studies addressing the association between free-living sleep and weight in children. Using data from the Gemini twin birth cohort, the objective of this thesis was to advance the understanding habitual sleep behaviour in early childhood and how it may operate to influence the development of adiposity. Study 1 identified the predictors and pathways to shorter sleep at age 15 months, demonstrating that multiple environmental factors are associated with shorter sleep in children, with several operating through a later bedtime. Studies 2 and 3 examined the association between sleep and energy intake at age 21 months. Study 2 identified a linear relationship between shorter sleep and energy intake, before an association between sleep and weight was observed. Study 3 demonstrated that shorter sleeping children consumed more calories at night only, and predominantly from milk drinks. Study 4 showed that shorter sleep was significantly associated with weight at age 5 years; and at this age shorter sleep was associated with higher food responsiveness, which could partly explain the association with a higher weight. Findings from study 5 highlight the role of the home environment, demonstrating a stronger association between sleep and weight among children living in higher risk home food environments. Overall, the results of this thesis highlight the importance of an early bedtime, and strongly suggest that shorter sleep in early life may lead to a greater propensity to over-consume. Shorter sleeping toddlers may consume more because of parents' inclination to feed to soothe at night, but changes in sensitivity to food stimuli may increase food intake and weight in older children with a greater autonomy over their eating behaviour. Implications, limitations and avenues for future research are discussed.

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Individual contribution

I played a lead role in developing the aims of this thesis, and in designing each of the five studies. However, invaluable input from my supervisors Dr Abi Fisher, Professor Jane Wardle, and Dr Clare Llewellyn helped me to refine my ideas throughout.

For Study 1, I was provided with the raw 15-month Gemini data (collected before I joined the team). The concept of the study was my own. I cleaned and coded all data included in the analyses, and performed the analyses myself.

For Study 2, I was provided with the raw energy intake and macronutrient data that had been coded by experts at the University of Cambridge Human Nutrition Research Unit. The concept of the study was developed in discussions with myself, Dr Abi Fisher, and Professor Jane Wardle. I conducted the literature review relevant to this work, wrote large sections of the original manuscript, and took over analysing and writing the manuscript when Dr Fisher went on sabbatical, independently carrying it through the peer-review process.

The same raw dietary data was used in Study 3. However, I recoded all this data (over 52,000 rows) to be time of day and eating occasion specific. The concept of the study was my own. I completed all analyses myself.

I was involved in the development of the 5-year questionnaire booklet, the data from which was used in Study 4. I independently collected all data from the 5-year questionnaire. I sent out questionnaires via mail and email (in a staggered fashion) to over 2300 families, as well are 3 subsequent email and written reminders. Hayley Syrad (a fellow PhD student) and I manually entered all the paper-booklets received (2 booklets from over 500 families each). With guidance from the Gemini data manager, I independently cleaned and coded all the parent and child data included in this wave of data collection. The concept of study 4 was my own, and I carried out all analyses and relevant background work myself.

For Study 5, I completed over 400 home environment interviews (lasting on average 40 minutes each). The development of the composite score formed the body of work for another Gemini PhD student, Dr Stephanie Schrempft. However, the concept of this study was my own, and I carried out all other analyses myself.

Chapter 1 Obesity, eating behaviour, and sleep

The first chapter will provide an overview of the key domains covered in this thesis: obesity, eating behaviour, and sleep.

1.1 Overweight and Obesity: definition, prevalence and impact

1.1.1 Definition

Overweight and obesity are the accumulation of excess body fat (adiposity), to the extent that health may be impaired. Adiposity is most often defined using body mass index (BMI), which is a measure of relative mass calculated according to an individual's height and weight (BMI = weight/height^2). Adults (>18 years) with a BMI of 25 or over are classified as overweight, and those with a BMI of 30 or over, obese.

In childhood, body fat percentage naturally fluctuates by age and sex (Lindsay et al., 2001). It rises sharply through infancy, falls during the preschool years, and then rises steadily again up until adulthood (Cole, Freeman, & Preece, 1995). In childhood, overweight and obesity are therefore defined according to age- and sex- specific standardised BMI scores, using percentiles rather than discrete BMI cut points. Overweight is classified as a BMI standard deviation score (BMI-SDS) between the 85th-95th percentile, and obesity as a BMI-SDS above the 95th percentile, for a given age and sex. In the UK, BMI centiles for children are derived from 1990 growth reference data (Cole et al., 1995).

1.1.2 Measuring adiposity: the utility of BMI

Although overweight and obesity are most commonly defined according to BMI (and BMI-SDS in children), BMI is a measure of excess weight (corrected for height) rather than a measure of excess fat. Many other methods are available that can index adiposity more precisely; dual-energy X-ray absorptiometry (DXA) for example is considered a gold standard measure. This method uses two X-ray beams, which are absorbed by the soft tissues of the body, to assess body composition in a way that can accurately distinguish between fat-mass and fat-free mass (Sopher et al., 2004). However, DXA scans require specialised and expensive equipment, and each scan emits a small amount of radiation, making their utility in large-scale epidemiological studies somewhat limited (Boeke et al., 2013). Due to its feasibility in large-scale studies, BMI is therefore often used as a proxy measure of adiposity in both children and adults. Importantly, although BMI does not directly measure body fatness, it correlates well with objective measures of body fat percentage, including DXA. For example, in a sample of 1,110 British children aged 6 to 11 years, fat mass obtained from DXA correlated highly with measured BMI ($r = 0.83$), as well as other frequently utilised indices of adiposity including waist circumference ($r = 0.86$) and skinfold thickness ($r = 0.79$) (Boeke et al., 2013). Therefore, when direct assessments of body fat are not practicable, BMI can provide an adequate proxy of adiposity for the purposes of population-based studies (Deurenberg et al., 2001; Pietrobelli et al., 1998).

1.1.3 Prevalence

The percentage of the adult population with a BMI in the overweight or obese range has steadily increased since the 1970's, and current rates of the disease are considered to be at epidemic proportions (Wang, McPherson, Marsh, Gortmaker, & Brown, 2011). In the UK alone, 67.1% of men and 57.2% of women are currently either overweight or obese (Health Survey for England, 2013). This can be compared with 57.6% of men and 48.6% of women in 1993 (Health Survey for England, 2013). This trend is projected to continue over the coming decades, with current forecasts estimating 11 million more obese adults in 2030 than 2010 (Wang et al., 2011). Although there is a trend for increasing obesity overall, prevalence is disproportionately higher among those from minority ethnic groups, lower socioeconomic backgrounds, and among those with lower levels of education (Wang & Beydoun, 2007).

Perhaps most concerning is the rising prevalence of overweight and obesity in children. Since the 1970's to the end of the 1990's the percentage of school-age children who are either overweight or obese has tripled (Wang & Lobstein, 2006), and while there is some indication that this rate of increase is stabilising, prevalence remains high (Ogden, Carroll, Kit, & Flegal, 2012). Objectively measured BMI-SDS data from over 1.1 million children in the 2014/15 National Child Measurement Programme found that 1 in 5 children aged 4-5 years, and 1 in 3 children aged 10-11 years in England are currently either overweight or obese (National Child Measurement program, 2015).

1.1.4 Impact

Overweight and obesity pose a considerable threat to population health. Adiposity is an established risk factor for a number of chronic diseases – most notably diabetes, cardiovascular diseases, and some cancers, including (but not limited to) cancer of the breast, colon, cervix and prostate (Pencina, D'Agostino, Larson, Massaro, & Vasan, 2009; Renehan, Tyson, Egger, Heller, & Zwahlen, 2008; Schienkiewitz, Schulze, Hoffmann, Kroke, & Boeing, 2006). By 2030, it is estimated that a continuing trend in obesity would present an additional 6-8 million cases of diabetes, 5-7 million cases of heart disease, and an additional 0.5 million cases of cancer in the UK alone (Wang et al., 2011). Excess weight also significantly contributes to many other debilitating disorders such as infertility, sleep apnoea, asthma, and osteoarthritis (Guh et al., 2009).

Although the detrimental health effects of adiposity can take years, if not decades to manifest, the rising prevalence of excess weight in early life poses a substantial health concern. This is because childhood obesity is itself associated with a number of adverse health outcomes that are precursors to serious longstanding illness, including hypertension (Salvadori et al., 2008), sleep apnoea (Arens & Muzumdar, 2010), and impaired glucose tolerance (Sinha et al., 2002). Indeed, once considered rare, Type 2 diabetes is now an emerging health issue among obese children and adolescents (Hannon, Rao, & Arslanian, 2005). Adiposity in early life has also been shown to predict future risk of disease independent of adult BMI (Tirosh et al., 2011). Furthermore, body weight tracks substantially over childhood, adolescence and adulthood (Guo & Chumlea, 1999); for example, one longitudinal cohort study of 2,610 American children found that those children who were overweight (> 95th BMI-SDS centile) at 2-5 years of age were over 4 times more likely to be overweight adults (Freedman et al., 2005). This is particularly concerning given that the number of years an individual lives with excess weight significantly contributes to overall morbidity and mortality risk (Abdullah et al., 2011).

The health impact of obesity, both in loss of life and impaired quality of life, is extensive. This in turn places a considerable economic burden on society, from increasing medical costs to losses in productivity. In most countries, spending on obesity-related healthcare accounts for between 2-6% of all healthcare costs (Wang et al., 2011). If trends continue, the UK alone will spend £2 billion a year in 2030 treating obesity and its related comorbidities (Wang et al., 2011).

The prevalence of multiple comorbidities as well as their social and economic costs emphasises the importance of understanding and treating obesity rather than simply its associated diseases. Moreover, the rising prevalence of excess weight in childhood underscores the importance of understanding the development of the disease in early life.

1.2 Aetiology

The aetiology of obesity is complex. Ultimately, adiposity results from a chronic energy imbalance determined by multiple interactions between environmental, genetic and behavioural factors operating to substantially increase energy intake and/or reduce energy expenditure (Hall et al., 2011). This section will primarily give an overview on what is known about environmental pressures driving energy imbalance, genetic influences on adiposity, and the role of appetitive traits, in order to understand why some individuals may be more prone to overconsumption and weight gain than others.

1.2.1 The obesogenic environment

Obesity has been described as a normal response to an abnormal environment (Swinburn et al., 2011). This is because the surge in rates of obesity in the 1980's largely coincided with shifts in the food and activity environment. At this time, technological advances allowed food to become increasingly processed, preserved, and mass-produced; the aftereffect being that convenient energy-dense food was freely available and cheap to consume (Hill & Peters, 1998; Swinburn et al., 2011). This effect has been exacerbated by a trend towards increasing portion sizes. In the United States for example, portion sizes for commonly consumed foods have been found to exceed government recommendations for a healthy portion by as much as 700% - in this case, for a chocolate chip cookie (Young & Nestle, 2002).

Changes in the food environment have been paralleled by substantial shifts in the activity environment. In particular, an increasingly urbanised, motorised and digitalised modern environment has meant that the energy expenditure needed for the activities of daily living has fallen considerably (Hallal et al., 2012; Swinburn et al., 2011). For example, over the last 50 years, it is estimated that occupation-related physical activity has decreased by the energy equivalence of more than 100 kcals per day (Church et al., 2011). The prominence of television and screen-based media in westernised societies has also meant that sedentary pastimes are now integral to modern life (Hill & Peters, 1998). Recent evidence suggests that children and adults spend on average 7.7 hours per day being sedentary (Matthews et al., 2008).

The 'obesogenic' environment is an umbrella term frequently used to describe the aforementioned surroundings, opportunities, and conditions of modern life that promote obesity (Swinburn & Egger, 2002). This can be further distinguished into macro and micro-level environments. Macro environments are broad, defined for example by how foods are taxed, supplied, or marketed nationally or internationally, while micro-level environments reflect direct environmental settings such as the home, school, workplace, or neighbourhood (Swinburn et al., 2011). In childhood, the home is an example of a micro-level environment thought to exert a strong influence on physical activity, dietary intake, and early life weight trajectories (Spurrier, Magarey, Golley, Curnow, & Sawyer, 2008; Strauss & Knight, 1999). For example, garden size and the amount of outdoor play equipment in the home has been associated with more outdoor play among children (Spurrier et al., 2008). Equally, the availability of certain food groups in the home such as sugar-sweetened beverages, or fruit and vegetables, has been positively associated with their consumption during childhood (Spurrier et al., 2008). Indeed, in one study that comprehensively assessed the home environment, homes evaluated as being 'healthier' were associated with lower BMI among low-income American school children aged 5-17 years (Pinard et al., 2014).

1.2.2 Genetic influences

Despite strong environmental pressures on adiposity, BMI is also known to be highly heritable. Twin studies indicate that 47-90% of the variation in BMI can be explained by genes (Elks et al., 2012). Multiple common genetic variants are thought to determine overall susceptibility to obesity, with each gene conferring a small effect (Yang et al., 2011). The fat mass and obesity- associated (FTO) gene was the first common obesity-associated genetic variant identified through genome-wide association studies (GWAS). FTO has the largest effect size of all common variants identified to date, but nevertheless has a relatively small effect size; it is associated with an average 3 kg weight difference between individuals with two lower risk alleles (TT) verses those with two higher risk alleles (AA) (Frayling et al., 2007). However, the most recent meta-analysis of GWAS established that there are now 97 common genetic variants that are robustly associated with BMI (Locke et al., 2015). Individuals with a greater number of higher risk variants for these 97 variants tend to fall at the higher end of the weight distribution, while those with more lower risk variants fall at the lower end.

1.2.3 Behavioural Susceptibility Theory

An increasingly obesogenic environment will place considerable pressure on the ability to maintain weight stability. However, in the face of this pressure not everyone becomes overweight or obese; substantial variability within the population exists despite increasing prevalence (Flegal & Troiano, 2000) indicating that some individuals are inherently more susceptible to environmental pressures than others (Blundell et al., 2005). Recent work has demonstrated that individual differences in susceptibility to the environment can be attributed to genetic variation.

Specifically, appetitive traits have been shown to be one of the pathways through which ‘obesity genes’ influence adiposity, and allow for genes to interact with the obesogenic environment – so-called Behavioural Susceptibility Theory (Carnell & Wardle, 2007). Appetitive traits reflect stable predispositions towards food and its consumption. The two traits that have been extensively studied in children are ‘satiety responsiveness’ and ‘food responsiveness’. Satiety responsiveness is the sensitivity to internal fullness signals, reflecting the homeostatic regulation of hunger, while food responsiveness is the tendency to want to eat when exposed to external food cues, reflecting hedonic processes in relation to food reward (Carnell & Wardle, 2007). The Behavioural Susceptibility Theory hypothesises that individuals who inherit a lower sensitivity to internal satiety mechanisms, and a greater responsiveness to external food cues, are more likely to overeat in response to the current obesogenic environment, and gain excessive weight (Llewellyn & Wardle, 2015).

The behavioural susceptibility theory has been supported by twin data and molecular genetic studies showing that appetitive characteristics have a strong genetic basis. For example, paediatric twin studies have demonstrated that both satiety responsiveness and food responsiveness are largely genetically determined, with approximately 70% and 60% of the variation respectively attributable to additive genetic effects (Wardle & Carnell, 2009; Llewllyn et al., 2010). The genetic underpinnings of these traits have also been supported by findings from molecular genetic studies. For example, the FTO gene has been shown to be involved in regulating satiety responsiveness, with AA homozygotes displaying significantly reduced satiety sensitivity in an eating in the absence of hunger task, compared to individuals carrying the low risk T allele (Wardle, Llewellyn, Sanderson, & Plomin, 2009; Wardle et al., 2008). In addition, a subsequent study showed that genetic risk of obesity quantified using 28 obesity-related genetic variants was associated with diminished satiety, and satiety sensitivity significantly mediated part of the association between genetic risk and adiposity, even when FTO was excluded (Llewellyn et al., 2014). This suggests that many of the obesity-associated genetic variants operate through effects on appetite.

Importantly, in paediatric samples, both satiety responsiveness and food responsiveness have been shown to vary in a continuous fashion with adiposity. Specifically, lower satiety responsiveness and higher food responsiveness are associated with greater adiposity in children (Wardle & Carnell, 2009; Webber, Hill, Saxton, Van Jaarsveld, & Wardle, 2009), indicating that these traits are not specific to those that are overweight, but rather vary quantitatively with weight (Carnell & Wardle, 2007). Indeed, both satiety responsiveness and food responsiveness have also been associated with obesity-promoting energy intake patterns (satiety responsiveness with bigger average meal size and food responsiveness with more frequent eating episodes), and increased overall energy intake (Syrad, Johnson, Wardle & LLewellyn, 2015). The two traits have also been associated with preference for less healthy foods; food responsiveness is associated with preference for non-core foods, which are those foods eaten for pleasure rather than as essential components a healthy diet (Fildes et al. 2015), and satiety responsiveness has been negatively associated with both fruit and vegetable liking and consumption (Fildes et al., 2015; Rosen et al., 2003). These associated behaviours help to explain why children with a predisposition towards the higher risk phenotypes are more prone to adiposity.

1.2.4 Homeostatic and hedonic regulation of food intake

At a physiological level, food intake is regulated by both homeostatic and hedonic pathways, and these pathways have a conceptual overlap with satiety responsiveness and food responsiveness respectively. Although the following two sections discuss homeostatic and hedonic pathways independently, the two systems are interrelated, and can both enhance and counteract one another to control food intake. For example, in a homeostatic state of negative energy balance reward processes may be upregulated, and reward processes may be dampened by homeostatic processes controlling satiation and satiety (Saper, Chou, & Elmquist, 2002).

1.2.4.1 Homeostatic regulation

The homeostatic control of food intake primarily involves the regulation of energy balance. This system is controlled by metabolic hormones acting on the hypothalamus to relay information on peripheral energy status (Saper et al., 2002). Leptin and ghrelin are two hormones that are central to homeostatic pathways. Leptin is an adipose-derived hormone that is expressed in relation to changes in fat mass, with higher serum levels observed alongside increases fat-mass (Klok, Jakobsdottir, & Drent, 2007). Leptin predominantly acts as a satiety hormone, operating to increase energy expenditure and supress food intake. A rare genetic mutation in the gene coding for leptin for example, causes leptin deficiency, which is a disorder characterised by hyperphasia and severe obesity (Montague et al., 1997). Acting in concert with leptin, ghrelin is a gut-derived hormone that increases in response to a negative energy balance, operating to stimulate hunger and food intake (Klok et al., 2007). For example, ghrelin levels rise shortly before a meal and fall sharply after feeding (Cummings et al., 2001), and exogenous administration of ghrelin to healthy participants has been shown to increase food consumed from an *ad libitum* buffet, relative to administration of a placebo (Wren et al., 2005). Therefore, by modulating feelings of satiety and hunger, the homeostatic system regulates food intake so as to maintain energy balance (Lutter & Nestler, 2009). However, an impaired physiological satiety response reflects an important risk factor for overconsumption, and is often observed in obese relative to healthy weight individuals (Van Name et al., 2015).

1.2.4.2 Hedonic regulation

Counter to the homeostatic energy balance system is the hedonic system, which can initiate food intake based on the perceived rewarding value of food, independent of physiological energy requirement. In this way, hedonic signals can override metabolic signals to promote energy intake that is in excess of energy requirements (Yu et al., 2015). This system is governed by activation in the mesolimbic dopamine pathways operating within the brain's reward system. The most consistently implicated regions involved in the hedonic control of feeding include the orbito-frontal cortex (OFC) and medial prefrontal cortex, the amygdala, insula, striatum, and anterior cingulate cortex (Morton, Cummings, Baskin, Barsh, & Schwartz, 2006). While hedonic feeding may have been adaptive in an environment where food was scarce, in a modern obesogenic environment hedonic processes have been strongly implicated in overconsumption and the development of obesity (Yu et al., 2015). Indeed, hyper-responsiveness in hedonic pathways is often observed in obese individuals. For example, relative to children of a healthy weight, obese children show elevated activity in brain regions implicated in reward processing in response to images palatable food, which also fails to diminish after food intake (Bruce et al., 2010).

1.3 Sleep

The next key domain covered in this thesis is sleep. Although the focus is predominantly nighttime sleep duration, sleep is a multidimensional construct. Therefore, the following sections will provide a brief overview of sleep, its regulation, and the health impact of insufficient sleep.

1.3.1 Definition

Sleep is a state characterised by altered consciousness; distinguished from wakefulness by a decreased responsiveness to external stimuli (Loomis, Harvey, & Hobart, 1937). Many physiological functions incur significant changes during sleep; in general, core body temperature is lowered, blood pressure falls, and breathing and heart rate slow (Khatri & Freis, 1967), although physiological changes depend on the sleep phase (section 1.3.2). Sleep is a fundamental biological requirement in all mammals – rats subjected to total sleep deprivation for example, die within a number of days (Everson, Bergmann, & Rechtschaffen, 1989).

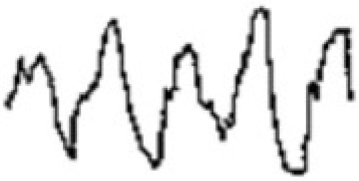
1.3.2 Sleep architecture

Electroencephalography (EEG) uses electrodes placed on the scalp to measure the electrical activity resulting from current flows between neurons in the brain, of which there are many billions (Lockley & Foster, 2012). The application of EEG measures during sleep has demonstrated that humans experience distinct types of sleep. These can be broadly categorised into non-rapid eye movement (NREM) and rapid eye movement (REM) sleep.

During sleep, the adult brain typically alternates between NREM and REM sleep in successive cycles lasting approximately 90-100 minutes each (Lockley & Foster, 2012). NREM sleep is accompanied by a stable physiological state characterised by a regular pattern of breathing, and slowed heart rate. There are 4 stages of NREM sleep, each identified by a distinct EEG wave type (Table 1.2.2). However, as there is no clear distinction between NREM stages 3 and 4, these are now more often called slow-wave sleep (SWS). SWS is the deepest sleep phase, and tends to occur most often during the first half of the night. SWS is thought to be essential for recovery and restoration, with the amount of time spent in this sleep phase correlating with prior sleep deprivation (Dijk, Brunner, Beersma, & Borbély, 1990).

Conversely, REM sleep is typically accompanied by a more variable physiological state, characterised by an inconsistent and higher breathing and heart rate, as well as increases in blood pressure. REM sleep most regularly occurs during the latter half of the night and is often followed by natural waking (Lockley & Foster, 2012). The time spent within each sleep phase, EEG wave type, and characteristics of each sleep phase are outlined in Table 1.3.2.

Table 1.3.2. Adult EEG patterns for different sleep phases[1]

Sleep stage	% Time asleep	EEG wave type	Unique characteristics
NREM stage 1	5%	Theta 	Drowsy sleep. Twitches and jerks in the limbs occur
NREM stage 2	45%	Theta, spindles, K-complexes 	Muscular activity decreases. Consciousness of the external environment disappears
NREM stage 3	12%	Delta, theta 	Slow-wave sleep. Deepests and most restrorative sleep Tissue growth and repair occurs Blood pressure drops, beathing slows, muscles relax
NREM stage 4	13%		
REM	25%	Beta 	EEG patterns are almost identical to those measured during wakefulness. Rapid eye movements. Skeletal muscles inhibited.

[1] Information in this table was adapted from *Sleep: A very short introduction* (Lockley & Foster, 2012) and from information provided by the National Sleep Foundation (https://sleepfoundation.org/)

1.3.3 The two process model of sleep-wake regulation

Although sleep is a multidimensional construct, the focus of this thesis is primarily sleep duration. The two-process model, first proposed by Borbely in 1982, provides a valuable framework from which to understand sleep-wake regulation in humans. The model proposes that there are two processes that interact to determine the duration and timing of sleep: sleep-wake homeostasis, and an internal 24-hour circadian rhythm (Achermann, 2004).

Sleep-wake homeostasis (called ‘Process S’ in the two-process model) is sleep-dependent and reflects the idea that the need for sleep accumulates throughout the day, such that the probability of falling asleep depends on how long an individual has been awake, and the probability of waking depends on how long an individual has been asleep.

The second part of the model, called ‘Process C’, describes the internal circadian pacemaker that regulates the timing of the sleep period and is sleep-independent. At a physiological level, Process C is located in the suprachiasmatic nucleus (SCN) of the hypothalamus. Process C operates as a circadian clock, generating a rhythm that is near to, but not exactly 24-hours. This near 24-hour rhythm in turn controls the timing of many physiological and behavioural functions, including sleep propensity, and is synchronised to the 24-hour day by external cues – most notably the light-dark cycle. This important feature of Process C enables the sleep-wake cycle to be coordinated with the environmental light-dark cycle. This can explain why it is generally easier to fall asleep at 12 midnight than 12 midday, and why sleeping in an adequately darkened room and limiting light exposure at night is an important aspect of good sleep hygiene (Mindell, Meltzer, Carskadon, & Chervin, 2009). Of course, natural variation in circadian rhythm can occur from person to person, as well as across the lifespan, in turn causing variation in both sleep duration and timing. For example, during adolescence and up until age 21 years, a natural delay in the circadian rhythm occurs. During this period in life, adolescents and young adults are inherently more inclined to go to bed later and wake up later in the day (Crowley, Acebo, & Carskadon, 2007).

Under normal circumstances Process S and Process C interact with one another to maintain wakefulness during the day, and an extended sleep period at night. Sleep pressure (Process S) builds the longer an individual stays awake, and abates during sleep. Process C counteracts Process S by increasing alertness during the day, serving to maintain wakefulness, and then falling at night, serving to aid sleep onset. In doing so, Process C normalises the timing of sleep so that it is initiated when sleep pressure surpasses an upper threshold at night, and waking is initiated when sleep pressure falls below a lower threshold in the morning (Crowley et al., 2007). An overview of the two-process model is shown in Figure 1.1. Of course, many factors (genetic and environmental) can interfere with this natural biological rhythm such that an adequate period of sleep at night is not achieved.

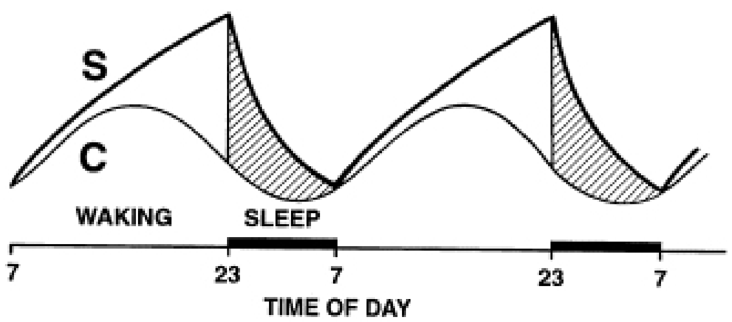


Figure 1.1. Overview of the two-process model of sleep-wake regulation. Process S rises during wakefulness and declines during sleep. The circadian Process C regulates the thresholds of sleep and wakefulness, synchronising the sleep-wake cycle with the environmental light-dark cycle. Shaded areas represent sleep period.

1.3.4 Genetic and environmental influences on sleep duration

Like adiposity, sleep duration is a complex phenotype that is determined by a dynamic interaction of genetic and environmental factors. Twin studies have demonstrated that the heritability of nighttime sleep duration in adults is between 31% and 55% (Watson et al., 2012). During early childhood (ages 6 to 48 months), 42-58% of the variance in sleep duration has been attributed to genetic factors (Touchette et al., 2013). Interestingly, there is some evidence that a critical window for environmental influence may occur between 15 and 18 months of age (Touchette et al., 2013). At this stage, two separate twin studies have shown nighttime sleep duration is predominantly influenced by the shared environment, which are those aspects of the environment that are completely shared by two twins in a family (Fisher, van Jaarsveld, Llewellyn, & Wardle, 2012; Touchette et al., 2013). For example, Touchette et al., 2013 explored the relative contributions of genetic and environmental factors on nighttime sleep duration in early childhood at 6, 18, 30, and 48 months of age. This study demonstrated that variance in nighttime sleep duration during early childhood is largely due to genetic factors with a critical environmental time-window influence at ~18 months (when 66% of the variance was attributed to the shared environment). This finding was supported by a heritability study in Gemini which demonstrated that nighttime sleep duration at 15 months of age was also predominantly influenced by the shared environment (48%: Fisher, van Jaarsveld, Llewellyn, & Wardle, 2012). Therefore, understanding the environmental exposures that influence sleep duration at a young age, particularly around 15-18 months of age, may be important.

In addition to genetic influences, many environmental exposures are known to influence sleep duration in early life. Increased screen time at night is one of the strongest correlates of shorter sleep in childhood (Garrison, Liekweg, & Christakis, 2011), perhaps because the light exposure and/or physiological arousal before bedtime displaces normal sleep onset by interfering with the internal circadian pacemaker (Cain & Gradisar, 2010), although the pathways are not yet clear.

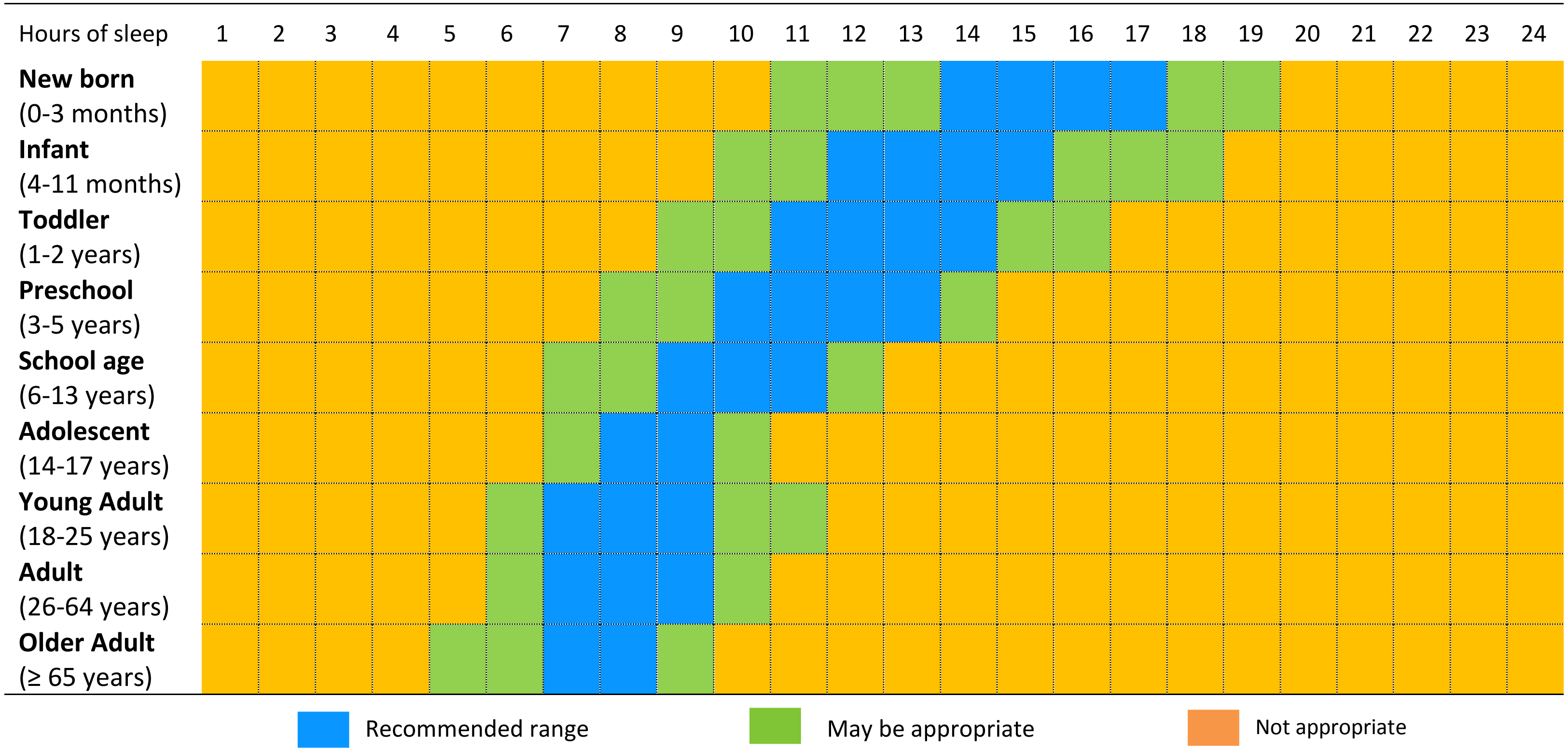
Sleep behaviour in childhood (as with adiposity) tends to cluster by socio-demographic factors such as ethnicity and maternal education. Specifically, children from ethnic minority families or more poorly educated backgrounds are significantly more likely to report shorter sleep at night (Blair et al., 2012). This is probably because ethnicity and maternal education shape attitudes and practices surrounding sleep. Children from minority ethnic backgrounds, for example, are more likely to report inconsistent bedtime routines and falling asleep with an adult present (McLaughlin et al., 2005; Mindell, Sadeh, Wiegand, How, & Goh, 2010). However, more research is needed to understand the pathways by which broad environmental influences such as ethnicity and socioeconomic status determine sleep duration in early life.

1.3.5 Defining short or insufficient sleep

Unlike adiposity, which has recognised thresholds to define ‘excess’, there are no clear definitions for what constitutes ‘short’ or ‘insufficient’ sleep. This is because sleep need is individualised and will depend on factors such as age, genetics, health status, and prior sleep debt. Indeed, defining what constitutes insufficient sleep will ultimately require experiments of sleep restriction and extension which include diverse health and behavioural outcomes (Magee, Iverson, Huang, & Caputi, 2008). This work is limited at present; however, one study by Sadeh et al. (2003) has demonstrated that extending or restricting nighttime sleep by 1 hour in 10 year old children had a significant impact on their neurobehavioral functioning, with sleep extension associated with more favourable outcomes on computerised cognitive tasks (Sadeh, Gruber, & Raviv, 2003). Similar experimental work is needed to clearly define what constitutes an adequate amount of sleep necessary to optimise health and wellbeing across the lifespan. Until this work is undertaken, population means provide the best available marker to define ‘adequate’ nighttime sleep.

Population means suggest sleep duration decreases steadily with increasing age; adults typically sleep around 7-9 hours, adolescents 8-10 hours, school-age children 10-11 hours, toddlers 12-14 hours, and infants 14-15 hours per day (Iglowstein, Jenni, Molinari, & Largo, 2003). Broad sleep recommendations from the National Sleep Foundation by age group are shown in Figure 1.2. Population-based studies of specific age groups can be used to provide more precise estimates at a given age. Figure 1.3 shows population estimates for nighttime sleep at multiple time points in early childhood. This work is used as a benchmark from which to compare the Gemini data presented in this thesis. Short sleep is then most often defined as shortened sleep relative to the population mean for a given age (Taveras et al., 2014).

Figure 1.2. Sleep recommendations per 24-hours across the lifespan by age group[2]



[2] Data adapted from the National Sleep Foundation (https://sleepfoundation.org/how-sleep-works/how-much-sleep-do-we-really-need)

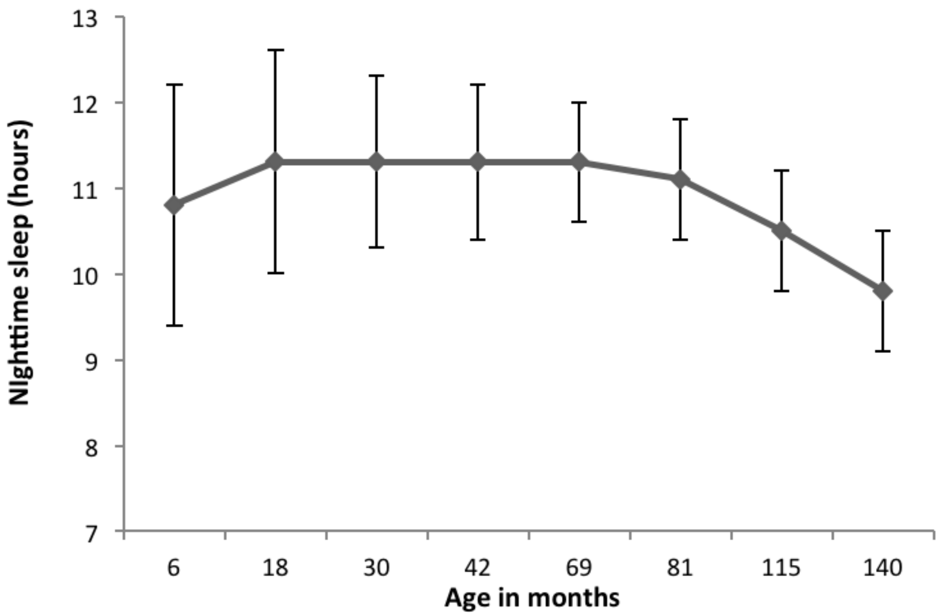


Figure 1.3. Nighttime sleep duration in a population sample of English children (ALSPAC) between 6 months and 11 years[3]

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[3] Data were adapted from Blair et al (2012), and were available for 7000 to 11 000 children at each time point. Error bars reflect standard error. Between 6 months and approximately 6 years of age (69 months) there was little change in mean reported nighttime sleep duration, remaining at 11.3 hours per night before declining slightly when the children reached school age (> 6 years).

1.3.6 Health impact of short sleep

Adequate sleep is a key pillar of good health. Epidemiological studies have consistently shown that insufficient sleep can to be detrimental to health and wellbeing across the lifespan. In adults, short sleep duration (sleeping <5-6 hours a night) has been shown to increase the risk of all-cause mortality (Cappuccio, D'Elia, Strazzullo, & Miller, 2010a), and has been associated with a number of serious diseases including, but not limited to, cardiovascular disease (Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011), breast and prostate cancer (Kakizaki, Inoue, et al., 2008; Kakizaki, Kuriyama, et al., 2008), and metabolic disorders including type 2 diabetes (Cappuccio, D'Elia, Strazzullo, & Miller, 2010b). Although the prevalence of non-communicable disease is generally low during childhood, insufficient sleep in early life has been associated with a number of disease precursors. Some evidence linking short sleep to disease outcomes related to obesity is reviewed below.

1.3.6.1 Short sleep and cardiovascular disease

The association between insufficient sleep and cardiovascular disease has been addressed in a number of large-scale epidemiological studies, with generally consistent findings. For example, a recent meta-analysis of 15 studies, including 474,684 adult participants, found that short sleep duration significantly increased the risk of developing or dying from cardiovascular disease (risk ratio: 1.48, 95% CI: 1.22 to 1.80, $P < 0.0001$), and stroke (risk ratio: 1.15, 95% CI: 1.00 to 1.31, $P = 0.047$). While the mechanisms are not yet understood, insufficient sleep has been associated with an increased risk of cardiovascular risk factors including artery calcifications (King et al., 2008), hypertension (Gottlieb et al., 2006), and metabolic dysfunction (Cappuccio, D'Elia, Strazzullo, & Miller, 2010b; Nedeltcheva, Imperial, & Penev, 2012). As will be discussed in Chapter 2, short sleep has also been consistently associated with obesity (Cappuccio et al., 2008), which is one of the strongest risk factors for cardiovascular morbidity and mortality (Abdullah et al., 2011).

1.3.6.2 Short sleep and metabolic dysfunction

In large epidemiological studies, short sleep has been associated with an increased risk of type 2 diabetes. A meta-analysis including 10 studies and a total of 107,756 participants found that adults sleeping less than 5-6 hours a night were at a significantly higher risk of developing type 2 diabetes compared to adults sleeping 7-8 hours a night (risk ratio: 1.28, 95% CI: 1.03 to 1.60, P = 0.024) (Cappuccio et al., 2010b).

Research has now begun to explore the underlying mechanisms through which sleep might impact metabolic function. For example, laboratory studies have shown that one week of sleep restriction (5 hours per night) in adults can reduce the effectiveness of insulin at lowering blood glucose levels (reduce insulin sensitivity), and impair the ability of glucose to regulate itself independent of insulin (reduce glucose tolerance) (Buxton et al., 2010). Importantly, both reduced insulin sensitivity and impaired glucose tolerance are metabolic disturbances that precede the onset of type 2 diabetes (Martin et al., 1992). Short sleep has also been implicated in the development of obesity, which is also one of the most important risk factors for the development of type 2 diabetes (Schienkiewitz et al., 2006).

1.3.6.3 The health impact of short sleep in early life

Shorter sleep in childhood has been described as a ‘noxious exposure’ that over time operates to substantially alter a child’s developmental trajectory, significantly increasing the risk of poor health (Beebe, 2011). Insufficient sleep in early life has been associated with many precursors to non-communicable disease. For example, among obese children, short sleep duration has been associated with higher levels of HOMA-IR, which is an index of insulin resistance, a precursor to type 2 diabetes (Flint et al., 2007). In one US study including over 14,000 healthy adolescents, each additional hour of sleep at baseline was associated with a 10-25% reduction in the odds of being diagnosed with hypercholesterolemia after 7 years (Gangwisch et al., 2010), which is the presence of excess cholesterol in the blood that can lead to atherosclerosis, a type of cardiovascular disease (Gordon et al., 1989).

Similar effects have been observed earlier in life. Cespedes et al. (2014) demonstrated that chronic insufficient sleep between infancy and age 7 was associated with increased 'metabolic risk', which in this study was derived as the mean of waist circumference, systolic blood pressure, HDL cholesterol, and HOMA-IR scores (Cespedes et al., 2014). However, this effect was found to be mediated by the impact of sleep on adiposity. Indeed, in both adults and children, there is now increasing attention on the role that shorter sleep may play in the development of overweight and obesity, which will be the focus of this thesis.

1.3.7 Sleep and obesity: intersecting epidemics

In modern society, sleep is often viewed as an indulgence, and good sleep is frequently sacrificed for social and professional demands. There is some evidence to suggest that rising levels of obesity have been paralleled by a secular decline in sleep duration (Iglowstein et al., 2003). Artificial lighting, the increasing availability of screen-based media and in adults, longer working hours and shift-work may all have contributed to declining nighttime sleep (Centers for Disease Control and Prevention, 2010). In the US, national surveys have reported a high prevalence (>35%) of short or insufficient sleep (Centre for Disease Control, 2010).

Interestingly, some research has suggested that the most pronounced decline in sleep duration over recent decades has occurred among children (Matricciani et al., 2012). A meta-regression of over 690,000 school-aged children in 20 countries found that sleep duration has declined by an average of 0.75 of a minute per year over the past 100 years (Matricciani, Olds, & Petkov, 2012). However, evidence supporting a secular decline in sleep duration among children has not been consistent. One recent review of the literature found that two studies reported an increase in sleep over time, six studies reported mixed results, three showed no change, and six reported a decrease in children's sleep duration over time, with much of the evidence evaluated as being of poor methodological quality (Matricciani, Olds, & Williams, 2011). Whether or not shorter sleep and obesity reflect parallel developments, there is now accumulating evidence to suggest that shorter sleep may be a risk factor for the development of overweight and obesity.

1.4 Summary

Overweight and obesity are the accumulation of excess body fat to the extent that health may be impaired. Excess weight results from a chronic energy imbalance determined by multiple interactions between environmental, genetic and behavioural factors. The Behavioural Susceptibility Theory proposes that genetically determined appetitive traits increase susceptibility to the obesogenic environment. The two most commonly studied traits are satiety responsiveness and food responsiveness, which reflect the homeostatic and hedonic control of food intake respectively. The second key domain covered in this thesis is nighttime sleep duration. Like adiposity, sleep duration is a complex phenotype that is determined by a dynamic interaction of genetic and environmental factors, but there are no clear definitions for what constitutes 'short sleep' so age-specific population means are most often used. However defined, adequate sleep is often considered a key pillar of good health, and there is some evidence to suggest that rising levels of obesity have been paralleled by a secular decline in sleep duration.

Chapter 2 Shorter sleep as a contributor to obesity

2.1 Shorter sleep and obesity risk: the evidence

2.1.1 Observational studies

A growing body of observational data suggests that shorter sleep is associated with weight gain and an increased incidence of overweight and obesity in both adults and children. Although the focus of this thesis is early childhood, much of the research in this area has been conducted with adults, so it is important to outline the evidence linking sleep and obesity risk in both adult and paediatric populations.

2.1.1.1 Adults

Several observational studies in adults have reported associations between short sleep and an increased risk of obesity. A quantitative estimate of this risk was reported in a meta-analysis of 18 studies including 603,519 adults aged 18-102 years. Pooled analyses showed that short sleep (<5 hours) increased the risk of obesity by 55% (pooled OR = 1.55; 95% CI: 1.43 to 1.68) (Cappuccio et al., 2008). Within the same study, a meta-analysis of regression coefficients demonstrated that there was a strong inverse linear relationship between decreasing sleep and increasing BMI. Specifically, each hour increase in sleep was associated with a −0.35 unit change in BMI (β = -0.35, 95% CI: -0.57 to -0.12; P = 0.002) (Cappuccio et al., 2008). For an average sized individual, this would be equivalent to approximately 1.4 kg in weight. While this is not substantial at an individual level, as discussed in Chapter 1, the aetiology of obesity is complex, and the effect of any single factor on body weight should be expected to be small. Moreover, at a population level, small differences have the capacity to shift the distribution of overweight and obesity, and are therefore of significant importance to public health (Hall et al., 2011).

The studies included in the above-mentioned meta-analysis were cross-sectional, so the possibility of reverse causation cannot be excluded. This is important given that obesity is associated with a number of comorbidities including asthma, arthritis, depression, and sleep apnoea, all which may disrupt normal sleep patterns (Ancoli-Israel, 2009; Foley, Ancoli-Israel, Britz, & Walsh, 2004). Crucially, prospective studies of adults have consistently shown that short sleep is associated with weight gain and future risk of obesity. In an analysis of 3,682 adults aged 32-49 in the National Health and Nutrition Examination Survey (NHANES), sleep duration at baseline was significantly linearly associated with weight gain after 9 years, with those adults sleeping ≤4 hours a night gaining on average 1.46 kg/m^2 (Gangwisch, Malaspina, Boden-Albala, & Heymsfield, 2005). Prospective studies have also shown that adults reporting improvements in sleep duration over a 6 year period (from ≤6 hours a day to 7-9 hours) have a lower fat-mass gain overtime than adults who maintain a short (≤6 hours) sleep duration (Chaput, Bouchard, & Tremblay, 2014).

It is important to note that a number of adult studies have reported a U-shaped association between sleep and weight (Taheri, Lin, Austin, Young, & Mignot, 2004). A recent systematic review of the evidence in adults, found that 6 of the 27 published studies reported an association whereby both short (<6 hours) and long (>8 hours) sleep were associated with a higher BMI (Patel & Hu, 2008). However, this evidence has come exclusively from cross-sectional analyses, and longer term follow-up has generally shown that only short sleep is associated weight gain (Patel, Malhotra, White, Gottlieb, & Hu, 2006). Any relationship between prolonged sleep and weight may therefore be due to reverse causation or residual confounding by physical and mental ill-health. For example, psychological symptoms like depression also show a U-shaped association with weight (de Wit, van Straten, van Herten, Penninx, & Cuijpers, 2009). Indeed, the association between sleep and weight is consistently linear in childhood when the prevalence of longstanding physical and metal ill-health is low (this evidence is discussed further in section 2.1.1.2). Understanding how longer sleep in adulthood associates with weight is however beyond the scope of this thesis. Moreover, the adverse effects of insufficient sleep may be more important to consider in today's society where there is some evidence for a secular decline in sleep duration (Bin, Marshall, & Glozier, 2012).

While there is consistent epidemiological evidence linking shorter sleep with increasing risk of obesity in adults, the strength of this relationship may dissipate with age (Gangwisch et al., 2005). For example, in the NHANES, the odds ratio for obesity in adults reporting very short sleep (≤4 hours) was 3.2, 1.8, and 1.7 for ages 32-49, 50-67, and 68-86 years respectively (Gangwisch et al., 2005). The increased mortality associated with obesity and age-related changes in sleep duration could be possible explanations for this age-effect. Broadly though, the relationship between short sleep and obesity risk is often discussed as being stronger at younger ages (Cappuccio et al., 2008).

2.1.1.2 Children

Short sleep has been consistently implicated as a risk factor for overweight and obesity in children. A meta-analysis of 11 cross-sectional studies (including 29,502 children aged 2-20 years) found a pooled odds ratio for obesity in those sleeping under 10 hours versus those sleeping more than 10 hours of 1.89 (95% CI: 1.46 to 2.43) (Cappuccio et al., 2008). The largest single paediatric study including 8,274 Japanese 6-7 year-olds found that children who slept less than 8 hours a night were almost three times more likely to be overweight than those who slept for more than 10 hours, after adjusting for age, sex, parental BMI, and outdoor playtime (Sekine et al., 2002). This relationship appears to be linear, particularly in younger children. A second meta-analysis published in the same year found a significant dose-response relationship between decreasing sleep duration and increasing BMI. Specifically, each hour increase in sleep was associated with a 9% decreased risk of overweight and obesity (pooled OR = 0.91; 95%CI: 0.84 to 1.00), and the linear trend was strongest in children under the age of 10 years (Chen, Beydoun, & Wang, 2008).

Shorter sleep has also been consistently associated with weight gain and risk of obesity in prospective analyses. For example, in a sample of 8,234 children in the UK, short nighttime sleep (<10.5 hours) at 3 years was independently associated with an increased risk of obesity (BMI-SDS ≥95th centile) at age 7 (OR = 1.45; 95% CI: 1.10 to 1.89)(Reilly et al., 2005). A study of 1,138 children in Canada found that those children who consistently slept <10 hours a night between 2.5 and 6 years of age were 4.2 times more likely to be overweight or obese than children who consistently slept for at least 11 hours during the same time period (OR = 4.2; 95% CI: 1.6 to 11.1) (Touchette et al., 2008). Indeed, the impact of shorter sleep on adiposity in early childhood may be cumulative. In a sample of over 1,000 families, chronic insufficient sleep between 6 months and 7 years of age was associated with greater total and trunk adiposity at age 7 years (measured using DXA) (Taveras et al., 2014). In this cohort, chronic short sleep was also associated with increased metabolic dysfunction at age 7, and these differences could be explained by greater fat mass among the shorter sleeping children (Cespedes et al., 2014). Consistently short sleep in early childhood may therefore have sustained effects on adiposity, and as a result, metabolic ill-health. It is important to note that most studies have found significant results even after controlling for potential confounding variables including socioeconomic status or parental education (Cespedes et al., 2014; Reilly et al., 2005; Taveras et al., 2014; Touchette et al., 2008), parental BMI (Cespedes et al., 2014; Taveras et al., 2014), birth weight (Touchette et al., 2008), and television viewing (Cespedes et al., 2014; Taveras et al., 2014; Touchette et al., 2008).

The majority of population-based studies investigating the association between sleep and obesity risk have used subjective measures of sleep duration (parent report), which may be prone to overestimate nighttime sleep (Owens et al., 2009). However, one of the few paediatric studies to objectively assess sleep with actigraphy found that each additional hour of nighttime sleep at age 3-5 years was associated with a reduction in BMI of -0.49 at age 7 years, corresponding to an average change of 0.7 kg. These differences could be explained by a higher fat mass as opposed to fat-free mass among shorter sleepers, as measured using DXA, and associations were retained after adjustment for multiple risk factors known to affect body weight in childhood (maternal education, ethnicity, TV viewing, physical activity, fruit/vegetable intake) (Carter, Taylor, Williams, & Taylor, 2011).

Other studies in children have found that shorter nighttime sleep predominantly affects abdominal adiposity. A study of 422 Canadian 6 year olds for example found that shorter sleep was independently associated with a higher waist circumference, independent of a broad set of potential confounding factors including age, parental BMI, parental education, family income, breakfast consumption, screen/media use, and sports participation (Chaput & Tremblay, 2007). This is important given that abdominal adiposity significantly contributes to metabolic abnormalities, and is associated with adverse inflammatory and cardio-metabolic alternations in children, even among those of a healthy weight (Galcheva, Iotova, Yotov, Bernasconi, & Street, 2011). If shorter sleep favours abdominal adiposity, then this could add to its deleterious effects.

Although shorter sleep has been shown to be more strongly associated with indices of adiposity in childhood, some published literature has suggested that early childhood (before the age of 5 years) reflects a 'critical period' when insufficient nighttime sleep may be an important risk factor for subsequent obesity (Bell & Zimmerman, 2010). In one longitudinal study of children aged 0-13 years at baseline, shorter nighttime sleep was strongly associated with risk of subsequent overweight and obesity at a 5-year follow-up in the younger cohort of children (aged 0-4 years at baseline), but not among the older children (aged 5-13 years at baseline) (Bell & Zimmerman, 2010). In contrast, one recent study found that chronic sleep curtailment throughout infancy, early and middle childhood was associated with greater total and trunk fat mass at age 7 years, without evidence for a 'critical period' (Taveras et al., 2014). However, longer-term follow-up into adolescence and adulthood is required to understand if early childhood does indeed reflect a 'critical period' for sleep curtailment.

After infancy and before the age of 5 years, young children sleep between 1 and 2 hours during the daytime (Iglowstein et al., 2003). Although daytime sleep represents a significant proportion of total sleep time that a child receives at this age, it has not been associated with an increased risk of obesity in prospective studies of young children (Bell & Zimmerman, 2010; Taveras, Rifas-Shiman, Oken, Gunderson, & Gillman, 2008). Daytime and nighttime sleep do have different predictors (Sadeh, Mindell, Luedtke, & Wiegand, 2009), and should therefore not be expected to have similar effects on weight in children. As such, the focus of the thesis is on nighttime sleep, although daytime sleep is generally considered and adjusted for in each study.

2.1.2 Experimental studies

A small number of experimental studies examining the impact of sleep curtailment on weight regulation have been conducted in adults, but there has been only one in children. However, this work is consistent in showing that short periods of sleep loss with *ad libitum* access to food are sufficient to generate weight gain in healthy individuals. For example, in a sample of 14 adult females studied at home, 4 nights of consecutively decreasing sleep (from 7 to 4 hours a night) led to a small but significant increase in body weight (0.4 kg) (Bosy-Westphal et al., 2008). Similar effects have been reported in laboratory-based studies. For example, in a sample of 16 healthy adults, restricting sleep to 5 hours a night for 5 days resulted in an average weight gain of just under 1 kg (Markwald et al., 2013). In a larger laboratory study including 225 adults, participants randomised to a sleep restriction condition (4 hours per night for 5 nights) gained approximately 1.4 kg over the study period, which was significantly more than control participants who gained on average 0.11 kg (Spaeth, Dinges, & Goel, 2013).

The one experimental paediatric study included 37 American children aged 8-11 years, 27% of whom were overweight or obese (Hart et al., 2013). Children were randomised to either increase or decrease their habitual nighttime sleep at home by 1.5 hours for 1 week (completing the alternate schedule the subsequent week), and wrist-worn actigraphy was used to measure sleep continuously. Relative to the increased sleep condition, when children decreased their sleep duration to 7 hours a night they gained on average 0.22 kg after 1 week (Hart et al., 2013). Although this weight change is relatively small, if sustained over the longer term, it does suggest that sleep duration could play an important role in the regulation of body weight in children.

Well-controlled experimental studies provide an important contribution to the literature as they are best able to delineate cause and effect in the sleep-weight relationship. However, inducing severe sleep restriction in an unnatural environment can limit the ecological validity of the findings. Furthermore, experimental studies cannot factor in the potential influence of habituation to chronic sleep curtailment, or help to understand whether small but sustained changes in sleep impact weight. Indeed, ethical issues associated with long-term sleep restriction, and the slow development of obesity, may mean this research is not possible. Consequently, it is important to consider the findings from short-term experimental studies alongside those of the epidemiological studies discussed in the previous section. When considered collectively, findings from epidemiological and experimental research provide compelling evidence that shorter sleep may be causally implicated in weight gain and adiposity in both children and adults.

2.2 Shorter sleep and energy balance

Energy balance describes the relationship between energy intake and expenditure, and provides a powerful framework for understanding the regulation of body weight (Hall et al., 2012). Under this framework, weight stability occurs when there is equilibrium between energy intake and expenditure (energy intake = expenditure). If shorter sleep causes weight gain it must therefore create an energy imbalance by reducing energy expenditure or increasing energy intake. There is evidence to demonstrate that sleep affects waking behaviours and physiological processes governing both sides of the energy balance equation, although effects on energy intake have been more consistently demonstrated. Figure 2.1 provides a schematic overview of the hypothesised relationship between short sleep and the components of energy balance discussed in the following sections.

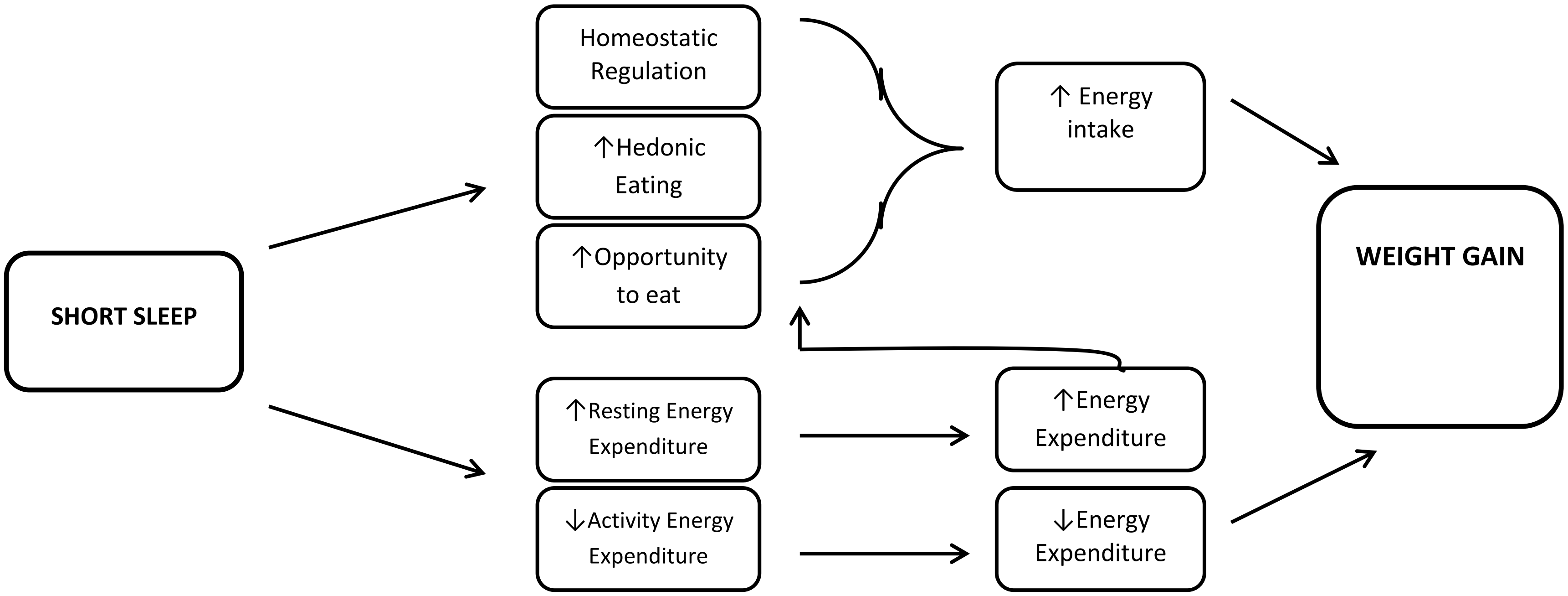

Figure 2.1. Schematic overview of the hypothesised pathways between short sleep and energy balance[4]

[4] Shorter sleep may create an energy imbalance by reducing energy expenditure (EE) and/or increasing energy intake (EI). Fatigue from insufficient sleep may reduce activity EE leading to reduced total EE. However, the energetic cost of extended wakefulness may also result in small increases in resting EE and therefore total EE. Because energy balance is a dynamic process, the energetic cost of wakefulness may encourage a series of behavioural and physiological changes aimed at increasing EI. Shorter sleep may directly encourage excess intake by deregulating the hormonal (homeostatic) regulation of appetite, increasing responsive to food stimuli leading to increased hedonic eating, or more simply by allowing for more time to eat.

2.2.1 Sleep and energy expenditure

Energy expenditure is a complex component of energy balance. Total energy expenditure consists of three main components: resting energy expenditure, thermic effect of food, and activity energy expenditure (Hall et al., 2011). Resting energy expenditure is the rate of energy required for core body functions at complete rest, and accounts for approximately 60% of total energy expenditure in a sedentary individual. The thermic effect of food is the amount of energy expenditure (above resting expenditure) required to digest and process food for use and storage, and accounts for about 10% of daily energy needs. Activity energy expenditure then refers to the amount of energy required during activity and includes both exercise energy expenditure and non-exercise activity thermogenesis, which is the energy required for all physical activities other than sport or gym based activities (Hall et al., 2012). Potentially, shorter sleep could exert a positive or negative effect on any of these components.

2.2.1.1 Activity energy expenditure

One of the most commonly discussed pathways by which shorter sleep might promote weight gain is through a reduction in activity energy expenditure caused by feelings of tiredness and fatigue (Chaput, 2013). There is some evidence to support this hypothesis, although findings have not been consistent. For example, in free-living adults, 2 nights of sleep restriction (4 hours a night) have been found to significantly increase feelings of tiredness, and decrease daytime physical activity, particularly from high-intensity activity, as measured via accelerometry (Schmid et al., 2009). Similar findings have been reported in habitually short sleeping adults. Using accelerometers to measure physical activity for 2-weeks, habitual short sleepers (<6 hours a night) were found to spend approximately 43 minutes less each day engaging in moderate to vigorous physical activity, and about 69 minutes more each day being sedentary (Booth et al., 2012). This reduction in activity energy expenditure is clinically relevant, equating to approximately 250 kcal per day for a 70 kg adult (Booth et al., 2012).

In contrast, some research in healthy adults has shown that sleep restriction *increases* physical activity, although effects have been moderate. For example, a single night of sleep restriction under free-living conditions has been shown to increase physical activity energy expenditure measured from accelerometers, but only by about 48 kcal per day (Brondel, Romer, Nougues, Touyarou, & Davenne, 2010). One population-based study found habitual short-sleeping adults exercised the most, despite also reporting higher BMI (Grandner, Jackson, Gerstner, & Knutson, 2013).

Among adolescents and children it is also unclear whether shorter sleep reduces activity energy expenditure. A study of 14,782 adolescents, found that self-reported daily physical activity of more than 60 minutes was associated with higher odds of sleeping at least 8 hours a night (OR = 1.67, 95%CI 1.46 to 1.91, $P < 0.005$), but no association was found between time spent in *vigorous* physical activity and nighttime sleep duration (Foti, Eaton, Lowry, & McKnight-Ely, 2011). In contrast, a repeated cross-sectional analysis of 234 children, measuring physical activity with accelerometers for 5 days at ages 3, 5, and 7 years, found that children who slept less at night were *more* active during the day and reported being less tired (Williams, Farmer, Taylor, & Taylor, 2014).

It is clear that studies investigating the relationship between sleep and physical activity have produced inconsistent findings. Generating conclusions from this limited work is made more difficult by the dissimilarities in methodology. For example, differences in how sleep and activity are measured – and within experimental paradigms – the number of nights of sleep restriction imposed, and whether activity is measured at specific times or continually throughout the study protocol, may all affect the findings. Indeed, one review of the literature has suggested that any change in activity associated with shorter sleep is more likely to be due to individual behavioural aspects than physiological changes (Klingenberg et al., 2012).

2.2.1.2 Resting energy expenditure and the thermic effect of food

A small number of studies have investigated whether experimentally induced sleep restriction causes intrinsic changes in metabolism. In general these have found that sleep restriction does not alter resting metabolic rate (Bosy-Westphal et al., 2008; St-Onge et al., 2011). For example, in a laboratory-based study of 11 healthy adults, sleep restriction (5.5 hours) was not associated with a change in energy expenditure relative to the rested condition (8.5 hours) (Nedeltcheva et al., 2009). Evidence regarding the influence of sleep restriction on the thermic effect of food is also limited and largely inconsistent, with studies showing either large increases (Bosy-Westphal et al., 2008), or decreases (Benedict et al., 2011) after short-term sleep restriction. Therefore, intrinsic changes in metabolism do not seem to contribute substantially to energy imbalance during sleep curtailment (Klingenberg et al., 2012).

2.2.1.3 Total energy expenditure

One of the functions of sleep may be to conserve energy. This is because daily energy expenditure is at its lowest during sleep; slowed breathing, lower body temperature and the lower amount of energy needed to maintain brain function and circulation means that resting energy expenditure is reduced by 20-30% during sleep (Penev, 2012). Furthermore, sleep imposed immobility means that both the thermic effect of food and activity related energy expenditure are largely absent during this time. It follows that shorter sleep (or extended wakefulness) will increase demands on total energy expenditure. Indeed, studies using a whole room calorimeter to measure changes in total energy expenditure have found that a single night of sleep deprivation increases nighttime energy expenditure by 32% (Jung et al., 2011). Similar results have been found with moderate levels of sleep restriction. Restricting sleep to 5 hours a night for 5 days has been shown to increase daily energy expenditure by 5% relative to baseline (9 hours sleep) (Markwald et al., 2013). This increase corresponds to the energy demands of extended wakefulness, and is clinically relevant. For a 70 kg adult a 5% increase in expenditure would equate to an extra 111 kcal per day; about the energy required to perform 24 minutes of aerobic exercise (Markwald et al., 2013). The energetic cost of prolonged wakefulness implies that shorter sleep should lead to weight *loss* – although all the evidence shows the contrary to be true.

2.2.2 Sleep and energy intake

Energy balance is a dynamic homeostatic process, and while it is useful to study each component individually, perturbations in energy expenditure are likely to result in compensatory changes in intake, and vice versa. One prevailing theory is that the energetic cost of increasing wakefulness encourages a series of behavioural and physiological changes aimed at increasing energy intake (Penev, 2012). In theory, these compensatory changes operate to restore energy balance; but in an obesogenic environment, where palatable energy-dense foods are abundant, overconsumption may follow. Indeed, evidence shows that increasing energy intake is the most plausible explanation for the weight gain associated with shorter sleep in adults, although studies in early childhood are lacking. A systematic search of the literature relating to sleep and eating behaviour in children was conducted (see Appendix A), however the literature retrieved was considered too diverse to adequately synthesise. Therefore, a narrative review of the literature relating to sleep and eating behaviour in both adults and children is provided in the following sections.

2.2.2.1 Energy intake: amount

In adult populations, intervention studies involving controlled sleep restriction have consistently demonstrated effects on food intake. Specifically, sleep restriction with a*d libitum* access to food has been shown to increase daily energy intake by between 10-25% (Brondel et al., 2010). Studies that have simultaneously measured energy expenditure have also found that this increase in energy intake is in excess of any rise in energy expenditure, and is sufficient to generate weight gain (Markwald et al., 2013). Furthermore, transitioning from short to normal sleep under controlled conditions has been shown to cause a reduction in food intake and promote weight loss (Markwald et al., 2013). A recent meta-analysis of 5 intervention studies found that sleep deprivation was one of the strongest lifestyle determinants of the drive to eat in adults, second only to alcohol consumption (Chapman, Benedict, Brooks, & Birgir, 2012).

To date, only one intervention study has been conducted in children. This study found that controlled changes in sleep duration in 37 children aged 8-11 years had a large effect on energy intake (Cohen’s d = 0.53), with children consuming 134 kcal per day more each day when sleep was restricted by 1.5 hours per night (Hart et al., 2013). This increase in energy intake is strikingly similar to that observed in adult studies of sleep restriction, reflecting approximately 8% of daily energy intake.

Experimental studies do not necessarily reflect habitual behaviours, and an important limitation is that they cannot factor in the potential influence of habituation to chronic sleep curtailment. Epidemiological studies therefore offer important additional sources of evidence, although few have specifically examined whether habitual short-sleepers consume more calories. In adults, the largest population-based study to date included 4,548 participants, and found that energy intake according to 24-hour recall varied significantly by sleep duration with self-reported short-sleepers (5-6 hours) consuming the most calories each day. However, the difference in energy intake between short and 'normal' sleepers (7-8 hours) was relatively small, averaging only 50 kcal per day (Grandner, Jackson, Gerstner, & Knutson, 2013b).

There are limited data in children and findings have not been consistent. Using two 24-hour recalls to measure habitual energy intake in 2,200 children aged 9-11 years, sleep duration was inversely associated with energy intake (Golley, Maher, Matricciani, & Olds, 2013). In contrast, within a smaller sample of children aged 10 years (n = 550), objectively measured sleep duration was not associated with energy intake assessed using 24-hour food recalls (Chaput et al., 2011). Similarly, in a sample of 802 Danish children aged 4-14 years, energy intake (assessed using 7-day diet diaries) was not significantly associated with sleep duration, although the association was in the hypothesised direction ($\beta = -0.015$, $P = 0.20$) (Hoppe et al., 2013). Given the effect size of the relationship when observed tends to small, it is possible these two smaller studies may have been underpowered to detect a relationship. However, it is also important to consider that self-reported dietary intake can be subject to recall bias, and under-reporting reflects a key limitation (Gemming, Jiang, Swinburn, Utter, & Mhurchu, 2014). Indeed, although 24-hour recalls are useful for large-scale studies, this method (particularly from a 1 or 2 day sample) may not precisely capture habitual dietary intake (Ma et al., 2010).

Taken together, experimental studies strongly implicate energy intake as the underlying pathway to the adiposity associated with shorter sleep, but more population-based research is needed to understand whether the effects are retained with habitual shortened sleep. Epidemiological research is particularly limited in young children, especially in those under the age of 5 years. This is important, not only because shorter sleep is more consistently associated with obesity risk early in life (Bell & Zimmerman, 2010; Cappuccio et al., 2008), but also because children self-direct their food choices to a much lesser extent than adults, so the impact of sleep restriction on energy intake cannot be assumed to be the same as that observed in adults, or even among older children. If energy intake does indeed drive weight gain in shorter sleepers, this also raises questions as to what may be driving overconsumption, and whether individuals who live in more obesogenic environments are more vulnerable to the effects of short sleep.

2.2.2.2 Energy intake: composition

Shorter sleep may also promote changes in the composition of dietary intake, encouraging a shift in preference towards energy-dense foods. Nutrient intake plays a central role in the development of chronic diseases (McCullough et al., 2002), so unfavourable changes could contribute not only to the development of adiposity, but also to its associated diseases.

Studies examining macronutrient composition in relation to sleep duration have predominantly shown effects on carbohydrate and fat intake. Specifically, periods of short-term sleep restriction in adults have been shown to encourage the consumption of carbohydrates, particularly those high in fat and sugar, such as desserts and sweets (Beebe et al., 2013; Markwald et al., 2013; Nedeltcheva, Imperial, & Penev, 2012). Intervention studies have also shown marked effects on fat intake. For example, St-Onge et al. (2011) found that healthy adults increased their dietary fat intake by 20 g and saturated fat intake by 9 g per day on average during 5 nights of sleep restriction. However, differences in dietary intake may be dependent on the type of food made available within the laboratory setting. In children, controlled changes in sleep duration obtained under free-living conditions have been found to alter energy intake without changing the relative macronutrient composition of the diet (Hart et al., 2013).

Some epidemiological data also suggest that shorter sleepers obtain a greater proportion of calories from fat or carbohydrate. In the Women's Health Initiative, objectively measured nighttime sleep in 459 post-menopausal women was negatively associated with percentage fat intake after adjustment for BMI (Grandner, Kripke, Naidoo, & Langer, 2010), and three adolescent studies have found that shorter sleepers have both higher carbohydrate and higher fat intake (Beebe et al., 2013; Bel et al., 2013; Weiss et al., 2010).

The largest study examining the relationship between sleep and dietary behaviour included data from 15,199 adults participating in the NHANES. This research demonstrated that, relative to average duration sleepers (7-8 hours), short sleeping adults (≤6 hours a night) consumed a greater proportion of their daily calories from snacks and sugar-sweetened beverages, but a smaller proportion from main meals (Kant & Graubard, 2014). Despite the observed differences in eating behaviour, differences in macronutrient composition between the sleep groups were small in magnitude, and no significant differences in total energy intake were observed.

Among children, shorter sleep has also been associated with unfavourable dietary patterns. Shorter sleeping children have been shown to consume a less varied, nutrient-poor, and energy-dense diet (Moreira et al., 2010; Westerlund, Ray, & Roos, 2009). Shorter sleep has also been inversely associated with fruit and vegetable intake, and positively associated with the consumption of fast foods (Collison et al., 2010; Golley et al., 2013; Tatone-Tokuda et al., 2012). Indeed, there is accumulating evidence in children that insufficient sleep may encourage the consumption of energy-dense foods, particularly those which are high in sugar. In a sample of 676 Danish school children aged 8-11 years, Kjeldsen et al. (2014) found that objectively measured nighttime sleep was negatively associated with energy density of the diet, added sugar consumption, and intake of sugar sweetened beverages (assessed using 7-day web-based diet diaries). In the same cohort, shorter sleep at baseline was associated with a higher intake of added sugar and sugar sweetened beverages at a 200 day follow-up, independent of potential confounding factors including pubertal status, ethnicity, parental education, and objectively measured physical activity and sedentary behaviour (Hjorth et al., 2014).

Importantly, the association between shorter sleep and dietary risk factors, including a higher intake of added sugar, could be a mediating factor in the observed association between sleep and metabolic health (Cespedes et al., 2014). Equally, a shift in food preferences favouring the consumption of energy-dense snack foods might also suggest that sleep loss preferentially impacts hedonic pathways underlying food intake (discussed further in section 2.4.2). However, more work is needed to understand the pathways by which shorter sleep might encourage changes in dietary composition.

2.2.2.3 Energy intake: distribution

Shorter sleepers may also be prone to consume a greater proportion of calories later in the day. In particular, experimental studies demonstrate that sleep restriction encourages the consumption of calories at night. For example, Spaeth et al. (2013) found that excess calorie intake in sleep restricted adults was specifically due to the consumption of additional calories between 10pm and 4am. In this study, nighttime energy intake alone accounted for between 14-22% of daily energy intake. Experimentally induced sleep loss has also been shown to preferentially affect nighttime energy intake in children. Hart et al. (2013) found that relative to all other behavioural and physiological endpoints, sleep loss had the greatest impact on nighttime energy intake in a group of 8-11 year old children (Cohen's d = 2.15). In the NHANES, short sleepers (≤6 hours) reported a higher proportion of calorie intake after 8pm, as well as a longer eating period (earlier time of first eating episode and later time of last eating episode) relative to average duration sleepers (Kant & Graubard, 2014).

The finding that sleep loss may impact the temporal distribution of energy intake is important as the timing of food intake has itself been implicated in weight regulation, with later eating times associated with higher BMI, poorer weight loss outcomes, and a higher risk of metabolic abnormalities (Corbalán-Tutau, Madrid, & Garaulet, 2012; Garaulet et al., 2013). As with changes in dietary composition, changes in the distribution of energy intake with decreasing sleep duration could not only contribute to the development of adiposity, but also to its associated diseases.

Epidemiological data in children support the idea that shorter sleep is associated with more irregular eating patterns including increased snacking, breakfast skipping and eating at unconventional hours (Tatone-Tokuda et al., 2012). However no studies have examined the temporal patterning of energy intake in relation to sleep duration in population samples of young children, and in particular no studies have investigated whether shorter sleeping children consume more at night. This is important because young children have limited autonomy over their feeding behaviour and food environment so they cannot be assumed to show the same temporal patterning of intake as adults. Furthermore, while experimental studies show clear effects of enforced sleep loss on nighttime energy intake, whether this also occurs under free-living conditions is not known.

2.3 Animal Studies

In the light of the observational data in humans, many rodent models have examined the metabolic impact of partial or total sleep deprivation. This work is consistent in showing that sleep deprivation induces hyperphagia and weight loss (Klingenberg, Sjödin, Holmbäck, Astrup, & Chaput, 2012). For example, Koban and Swinson (2005) demonstrated that rodents exposed to 20 days of sleep deprivation increased their food consumption by 220% during a 20 day study period, and lost 11% of their initial body mass. During the recovery phase however, both food intake and body weight returned to baseline levels. In rodent models, weight loss in the context of sleep-induced hyperphagia may be caused by elevated energy expenditure driven by the energetic need required to support wakefulness (Hipólide et al., 2006). This may be exacerbated by the methods used to promote wakefulness in rodents, which often involve excessive activity (e.g. wheel running). However rodent models also consistently show that sleep deprivation disrupts thermoregulation resulting in an upregulation of brown adipose tissue activation to offset a drop in body temperature. Brown adipose tissue is more abundant in rodents than humans, which could help to explain the discrepant findings in weight change between human and rodent studies. Recently, animal studies have shown that light exposure at night increases bodyweight and disrupts metabolic functioning (Fronken et al, 2013), perhaps pointing to another mechanism through which sleep restriction could promote weight gain. While animal models provide insight into the reciprocal links between sleep and energy homeostasis, physiological differences between species, as well as the extreme paradigms of sleep deprivation used in animal models may account for some discrepant findings and may mean that this work cannot be reliably extrapolated to humans.

2.4 Mechanisms by which insufficient sleep may increase energy intake

Research has begun to elucidate the mechanisms by which shorter sleep might operate to alter the amount, composition and distribution of energy intake in humans. The processes that have been most strongly implicated in driving these changes are the deregulation of appetite hormones, enhanced reward sensitivity, and increased time available for consumption. Although each mechanism has been studied and will be discussed independently, they are not necessarily mutually exclusive and may operate concurrently to promote excess energy intake in shorter sleepers.

2.4.1 Homeostatic processes

At a physiological level, hunger and satiety are regulated through the secretion of hormones indicative of energy status (Chapter 1, section 1.2.4). A dominant hypothesis within the literature is that sleep restriction disrupts hormonal signals in a way that enhances hunger and promotes energy intake (Copinschi, Leproult, & Spiegel, 2014). In particular sleep restriction has been shown to disrupt the regulation of leptin (an adipocyte-derived hormone that supresses appetite) and ghrelin (a gut-derived hormone that stimulates appetite) (Van Cauter et al., 2007). The homeostatic regulation of food intake was introduced and discussed in Chapter 1, section 1.2.4.

The seminal work by Spiegel et al. (2004) was the first to demonstrate that periods of short-term sleep restriction can deregulate appetite hormones. In this randomised controlled crossover study, 12 healthy young men were exposed to 2 nights of 4 hours, and 10 hours total sleep time while being administered standardised glucose infusions to avoid meal-related fluctuations in appetite hormones. Spiegel et al. (2004) found that, relative to the sleep extension phase, 2 nights of sleep restriction caused leptin levels to decrease by 18%, and ghrelin levels to increase by 28%. Furthermore, participants' subjective ratings of hunger increased by 34%, were highest for energy-dense high carbohydrate foods, and correlated significantly with changes in the leptin to ghrelin ratio – strongly suggesting that sleep restriction enhances hunger via an up-regulation in appetite hormones (Speigel, Tasali, Penev, & Van Cauter, 2004).

A key methodological consideration in the work by Spiegel et al. (2004) however, was that calorie intake was kept constant by providing participants with time- and weight-standardised levels of intravenous glucose (5 g per kg of body weight every 24 hours). Given that being awake, rather than asleep, incurs an energetic cost (section 2.2.1), participants would have expended more energy in the sleep-restricted phase relative to the rested phase, resulting in a negative energy balance. Just as energy intake causes fluctuations in appetite hormones aimed at supressing hunger, being in a negative energy balance causes fluctuations aimed at stimulating energy intake (Perry & Wang, 2012). Therefore, decreasing leptin and increasing ghrelin could characterise an appropriate counter-regulatory response to an energy deficit – although it is also possible that sleep restriction enhances normal regulatory responses to disproportionately increase hunger or decrease satiety.

Importantly, not all studies have found that sleep restriction disrupts metabolic hormones. Sleep restriction protocols that provide participants with *ad libitum* access to food have shown that participants' increase their energy intake despite normal regulatory changes in appetite hormones signalling a positive balance (increased leptin, decreased ghrelin) (Markwald et al., 2013). This suggests that in the context of free access to palatable food, hedonic (reward-driven) as opposed to homeostatic factors may drive excess energy intake during sleep restriction. This situation might better reflect what occurs under free-living conditions in habitual short sleepers (Chaput, 2013).

Appetite hormones are acutely sensitive to perturbations in energy balance (Perry & Wang, 2012). As such, isolating the effect of sleep loss on the regulation of these hormones is inherently difficult. Indeed, the conflicting results detailed above can probably be explained, at least to some extent, by differences in the energy balance of study participants. However, virtually all metabolic and endocrine systems are regulated by the 24-hour circadian rhythm (Copinschi et al., 2014), so the disruption of appetite hormones remains a viable hypothesis by which short sleep might promote energy intake and facilitate weight gain. Moreover, experimental studies do not provide information on the effects of long-term sleep curtailment, and more work is needed to understand the regulation of appetite hormones in habitually short sleepers. Interestingly, some research has shown that routinely short sleepers (<5 hours) have low levels of leptin and elevated levels ghrelin in morning fasted blood samples compared to adults reporting habitual sleep of at least 8 hours a night, independent of BMI (Taheri et al., 2004).

2.4.2 Hedonic processes

Acting in concert with the homeostatic energy balance system is the hedonic system. In the context of energy abundance, hedonic signals are thought to override inhibitory metabolic signals to stimulate overeating (Yu et al., 2015). The hedonic system is governed by pleasure, such that food intake is based on the perceived rewarding value of food, rather than physiological energy requirement. As discussed in Chapter 1 (section 1.2.4) perturbations in hedonic processes have been implicated in the development of obesity (Yu et al., 2015). Importantly, there is also emerging evidence that sleep loss may alter hedonic processes underlying the drive to eat (Chaput & St-Onge, 2014).

Studies using functional magnetic resonance imaging (fMRI) have shown that sleep restriction changes brain activity in response to food stimuli in a way that may operate to significantly increase food intake. During a food desirability task for example, Greer et al. (2013) found that sleep restriction diminished brain activity in higher-order cortical regions involved in food evaluation (anterior cingulate, anterior insula, and orbital frontal cortices), and increased activity in sub-cortical regions involved in signalling food salience (amygdala). This shift in brain activity was associated with an increased desire for energy-dense foods, and occurred independently of BMI. Additional food was provided during sleep loss to offset the energetic cost of extended wakefulness, so participants did not report feeling more hungry during this time. The observed differences in food processing and desirability cannot therefore be accounted for by a discrepancy in energy needs or perceived hunger, but seem to be a direct implication of sleep loss.

The change in the profile of brain activity observed by Greer et al. (2013) probably results in the inadequate evaluation of food stimuli, and the increased salience of energy-dense foods. In the context of unlimited eating opportunities, these adaptations are likely to contribute to excess energy intake detailed in the previous section. Indeed, two other studies have recently shown that sleep restriction enhances neuronal responses underlying the rewarding value of food (Benedict et al., 2012; St-Onge et al., 2012). In a modern obesogenic environment, the possibility that sleep loss makes food stimuli both more salient and rewarding provides a compelling explanation as to how insufficient sleep may motivate overconsumption, particularly from energy-dense foods.

As discussed earlier in this chapter (section 2.2.2.2), studies on dietary intake have shown that shorter sleeping children have more unfavourable dietary patterns, in particular a greater preference for, and intake of, energy-dense foods. Importantly, such patterns of consumption are suggestive of 'hedonic overeating', where eating occurs in response to palatable food cues rather than physiologic need (Yu et al., 2015). However, most studies to date have investigated *acute* effects of sleep deprivation on hedonic eating or stimulus processing; there have been few studies examining habitual eating behaviour in relation to sleep under free living conditions. In one small study of 56 children aged 5-12 years, shorter sleep was associated with lower scores on a scale of 'external eating', a construct that reflects the child's responsiveness to external food cues (Burt, Dube, Thibault, & Gruber, 2013). Among adults, one study has also shown that a tendency to disinhibited eating moderates the association between sleep and BMI, with a stronger relationship among adults who had higher disinhibited eating (Chaput, Després, Bouchard, & Tremblay, 2011). Again, disinhibited eating has some overlap with external eating in that it reflects the propensity to eat opportunistically within an obesogenic environment (Bryant, King, & Blundell, 2008). To better understand whether hedonic processes underlie excess food intake among shorter sleepers however, more work is needed to examine the relationship between habitual sleep, hedonic eating, and weight.

2.4.3 Time available for consumption

In addition to the physiologic effects of sleep on appetite and reward sensitivity, shorter sleepers may eat more simply because they are awake for longer. Specifically, shorter sleep parallels longer periods of wakefulness and exposure to the obesogenic environment, which allows more opportunities for consumption.

This idea is supported mainly by experimental data from children and adults demonstrating that recurrent sleep restriction increases energy intake predominantly during the hours of prolonged wakefulness (Hart et al., 2013; Nedeltcheva et al., 2009; Spaeth, Dinges, & Goel, 2013). For example, Hart et al. (2013) restricted children's sleep by delaying their bedtime under free-living conditions, and found that 76% of the extra calories consumed were eaten during the 3 additional hours children were awake at night. Spaeth et al. (2013) found that extended wakefulness alone (following a period of habitual sleep) was sufficient to increase intake by 670 kcal between 10pm and 4am. However, in the NHANES, habitual shorter sleeping adults reported an earlier time of first eating episode as well as a later time of last eating episode, without differences in total energy intake between the sleep groups (Grandner, Chakravorty, Perlis, Oliver, & Gurubhagavatula, 2014). Under-reporting however remains a limitation of self-reported dietary intake, and may be more prevalent among those who are overweight or obese, so caution should be taken when interpreting this data (Gemming et al., 2014).

Nevertheless, experimental work provides some evidence that prolonged wakefulness may facilitate the consumption of excess energy; however, it may be that increasing wakefulness at night (as opposed to in the morning) may be most critical at promoting overconsumption. Laboratory-based studies which have restricted sleep by simultaneously delaying bedtime and advancing morning wake time have found this promotes excess energy intake during the additional time spent awake at night, but not during the morning (Markwald et al., 2013). In fact, sleep-restricted participants have been shown to consume *less* in the morning when morning wake times were advanced (Markwald et al., 2013). The idea that being awake for longer at night may facilitate overconsumption is also supported by studies demonstrating that people who tend to go to bed later and wake up later also report a higher energy intake and poorer diet, independent of nighttime sleep duration (Baron, Reid, Kern, & Zee, 2011a; Sato-Mito et al., 2011).

The nighttime period may reflect a particularly vulnerable period for overconsumption as there is some evidence that hunger levels are endogenously higher during this time (Scheer, Morris, & Shea, 2013). Furthermore, time awake at night is generally spent engaging in sedentary activities (e.g. TV viewing) which themselves may promote energy intake (Harris, Bargh, & Brownell, 2009). Therefore, having more time to eat may contribute to excess energy intake in shorter sleepers, but having more time to eat *at night* may be pivotal in promoting passive overconsumption. This could also help explain why shorter sleepers often report an altered distribution of energy intake, with higher energy intake at night.

Although research is beginning to unravel the mechanisms driving energy intake in shorter sleeping adults, more work in young children is needed to understand the processes by which shorter sleep might contribute to weight gain and an increased risk of obesity.

2.5 Progression Hypothesis

The basic model guiding the work in this thesis is that the relationship observed between sleep duration and obesity in adults is established by a pattern of sleep duration and food intake in infants that is believed to persist. This can be referred to as the 'progression' hypothesis. In order to test the progression view it was hypothesised that there would be an association between sleep duration at 15 months and a measure of BMI at 5 years. A test of this hypothesis revealed no significant association, although the association was in the hypothesised direction (β = -0.09, 95% CI -0.2 to 0.03, P = 0.14). The absence of any significant relationship weakens the idea that sleep duration in infancy can exert a lasting effect on BMI. However the very small sample size with available BMI data at 5 years (n=536) may mean there was not adequate power to detect an effect. Furthermore, the best way to test the longitudinal relationship between sleep duration and weight would be to examine whether changes in sleep overtime correlate with changes in weight. There were not enough assessments of sleep in the Gemini data to do this, but previous studies have shown effects in samples of young children (Taveras et al., 2014, discussed in section 2.1.1.2). Nevertheless, it is important to present negative outcomes, and this finding should be taken into account when evaluating the overall strength of the theory of sleep duration and obesity. This finding may suggest that any sleep-obesity relationship is not as strong as many research reports suggest.

2.6 Summary

Short sleep has been strongly implicated as a risk factor for overweight and obesity in both children and adults, although the association appears stronger at younger ages. Increasing energy intake, rather than decreasing energy expenditure, is the most prevailing causal explanation for the weight gain associated with short sleep. Periods of controlled sleep restriction increase energy intake, particularly at night and from high fat and carbohydrate foods. Excess energy intake during sleep loss may be driven by a deregulation in the neuroendocrine control of appetite, altered reward sensitivity, or more simply because shorter sleepers have more time to consume (particularly at night). However, it is not yet clear what pathways are involved in driving weight gain in habitually short sleeping young children, who may be more vulnerable to the effects of insufficient sleep, but who also have limited dietary autonomy.

Chapter 3 Aims of the thesis

Chapter 1 gave an overview of the key domains covered in this thesis, while Chapter 2 outlined the evidence demonstrating that shorter sleep is associated with an increased risk of obesity, and the association appears stronger at younger ages. The mechanisms by which shorter sleep may contribute to adiposity are beginning to be unravelled, but there is limited research in young children. Therefore, the overarching aim of this thesis is to better understand habitual sleep behaviour in early childhood, and how it may operate to influence the development of adiposity in early life. The specific aims of the thesis, and the studies addressing each aim, are detailed below. These are:

(i) To identify the predictors and pathways to shorter sleep in early life.

- **Study 1** identified the predictors of shorter sleep at 16 months of age, and tested whether these operate primarily through sleep initiation (bedtime) or sleep termination (wake time).

  A version of this chapter has been published as: McDonald, L., Wardle, J., van Jaarsveld, C. H. M., Llewellyn, C., Fisher, A. (2014). Predictors of shorter sleep in early childhood. *Sleep Medicine*, *15*, 536-540.

(ii) To examine the relationship between sleep and eating behaviour in early childhood, and how this may influence the development of adiposity

- **Study 2** examined whether shorter sleep at 16 months is associated with a higher energy intake at 21 months, testing the hypothesis that shorter sleep would be related to higher energy intake in early childhood

  A version of this chapter has been published as: Fisher A., McDonald L., van Jaarsveld C.H.M., Llewellyn C., Schrempft S., Wardle J. (2014). Sleep and energy intake in early childhood. *International Journal of Obesity, 38*, 926–929.

- **Study 3** examined the temporal patterning of energy intake in relation to sleep duration testing the hypothesis that energy intake at 21 months would be specifically higher in the evening in shorter-sleeping children.

  A version of this chapter has been published as: McDonald, L., Wardle, J., Johnson, L., van Jaarsveld, C. H. M., Llewellyn, C., Fisher, A. (2014). Sleep and nighttime energy consumption in early childhood: a population-based cohort study. *Pediatric Obesity*, *10*, 454-460.

- **Study 4** tested the hypothesis that shorter-sleep is associated with hedonic eating, and explored the mediation of the relationship between sleep and weight by food responsiveness (an index of hedonic eating) at age 5 years.

  A version of this study has been published as: McDonald, L., Wardle, J., Llewellyn, C., Fisher, A. (2015). Nighttime sleep duration and hedonic eating in childhood. *International Journal of Obesity*, 39**,** 1463–1466. This was the first original research manuscript accepted to the *International Journal of Obesity* without corrections.

(iii) To examine the interplay between sleep duration and the home environment, and how this may influence the development of adiposity in childhood.

- **Study 5** examined whether the degree of risk within the home food environment moderated the cross-sectional association between sleep and BMI-SDS at age 5 years, testing the hypothesis that a stronger relationship between sleep and weight would be observed among children living in a higher risk home food environment.

Chapter 4 Methods

4.1 Overview of Gemini – a UK twin birth cohort

Each of the studies presented in this thesis use data from the Gemini study. The Gemini study is a population-based cohort of twins in the UK set up to examine the determinants of early childhood weight trajectories, with a specific focus on behavioural mechanisms (van Jaarsveld, Johnson, Llewellyn, & Wardle, 2010). The study was established by Professor Jane Wardle in 2007 to (i) understand the genetic and environmental contributions to weight gain in early childhood, (ii) identify modifiable determinants of excessive weight gain, and (iii) to create a resource of data on early childhood exposures that may determine long-term health (van Jaarsveld et al., 2010).

4.2 Sample: Recruitment and representativeness

In January 2008, the Office of National Statistics contacted all families with twins born in the UK between March and December 2007 (n = 6,754). Baseline questionnaires were sent to families that were willing to be contacted (n = 3,435), and 2,402 families agreed to participate by returning the questionnaires (36% of all live twin births, and 70% of those willing to be approached). All parents provided informed written consent with baseline questionnaires. The study was granted ethnical approval by the University College London Committee of non-National Health Service Human Research, and all aspects of data collection and storage were in compliance with the standards specified by this agency.

The geographic distribution of Gemini families reflects the UK population density. The sample is also comparable to UK twin statistics for sex, gestational age, and birth weight (data shown in Table 4.1). However, in common with other population-based cohorts, ethnic minority families and lower socioeconomic groups are somewhat under-represented. Furthermore, Gemini mothers are slightly older, and slightly healthier than population averages with respect to diet, smoking rates and BMI. Baseline characteristics of the Gemini mothers relative to national statistics are presented in Table 4.2.

Table 4.1. Characteristics of Gemini twins compared to National twin statistics[a]. Data are n (%) unless stated otherwise[5]

	Gemini Cohort (n = 2402 families; n = 4804 twins)	**National twin statistics [a]**
Gestational age, mean (SD)	36.20 (2.48)	37
Birth weight, mean (SD)	2.46 (0.54)	2.50
Pre-term (<37 weeks)	1045 (43.5)	40
Sex of twin pair		
Male	785 (32.7)	32.1
Female	801 (33.3)	32.8
Opposite sex	816 (34.0)	35.1

[5] Information presented in this table was adapted with permission from the Gemini development paper, van Jaarsveld et al. (2010)

Table 4.2. Baseline characteristics of Gemini mothers compared to national statistics[6]

	Gemini cohort (n = 2402 families; n = 4804 twins)	National statistics
	Mean (SD)	*Mean*
Age at twin's birth in years		
Mother	32.95 (5.19)	29.5[a]
BMI in kg/m^2, [b]		
Mother	25.10 (4.76)	26.9[d]
Gestational age (weeks)	36.20	39.25
Birth weight (grams)	2426	3352
	n (%)	*%*
Mother's ethnicity		
White	92.9 (2231)	88.2 [a]
Non-White	7.0 (169)	11.8 [a]
Not known	0.1 (2)	-
Mother's education		
Low	518(21.6)	31[c]
Medium	878 (36.6)	40 [c]
High	1006 (41.9)	29 [c]
Five portions of fruit or vegetables a day (at least)		
Mother	790 (32.9)	31.0 [d]
Current smoker		
Mother	306 (12.7)	21.0 [d]

[a] Office for National Statistics (2006). ONS Population report for England and Wales. Statistics correspond to parents with life births in 2006

[b] BMI calculated from self-reported height and weight

[c] Labour Force Survey, Quarter 2, 2008. Statistics are based on males and females of working age (16-64 and 16-59 respectively)

[d] Health Survey for England 2007 Volume 1. Health lifestyles: knowledge, attitudes and behavior. Ed R. Craig & N. Shelton. The health and social care Information Centre, 2008.

[6] Information presented in this table was adapted with permission from the Gemini development paper, van Jaarsveld et al. (2010)

4.3 Data Collection

From its initiation in 2007, the Gemini study has collected extensive data on weight, and factors known to influence weight in early life. The completed work in this thesis uses data from measures of sleep, dietary intake, eating behaviour, and the home environment, as well as anthropometric data. Data has been collected at multiple time-points, primarily using paper-based or online questionnaires. Table 4.3 provides a schematic overview of the each assessment point in Gemini with data points included in this thesis. This table also provides information on the year of data collection, and the response rate for each questionnaire. A copy of all questionnaires with data used in this thesis can be found in the Appendix.

4.3.1 Attrition

The response rates for each wave of data collection are given in Table 4.3. There has some attrition in the Gemini study over the 6-year study period, with 45% of baseline families completing the most recent wave of data collection (the 5 year questionnaire). Attrition should be expected in any longitudinal population-based study, but limits the generalisability of the findings presented in this thesis to some extent. In particular, mothers who completed each wave of data collection tended to be older and more highly educated, and were more likely to be from a white ethnic background. This issue is generally considered in each study chapter, and the differences between responders and non-responders are reported. However, this is an important limitation with the study sample and should be carefully considered when interpreting the findings. This issue is discussed further in the General Discussion.

4.3.2 Personal contribution

I have personally been a part of the Gemini team since 2011. During this time I have been involved in all aspects of running the cohort including data collection, data entry, data cleaning, maintenance of the database, developing the annual newsletter, and providing a regular point of contact for participating families. I recoded the 21-month dietary data to be time-of-day specific. I also completed over 400 home environment interviews and coordinated the mail-out, processing, data entry, data cleaning, and data coding for the 5-year questionnaires. Throughout my thesis I have also helped run the Gemini emails, have been responsible for ensuring contact details in the database are correct and up-to-date, and personally send out the measurement reminders to all participating families every 3 months.

Table 4.3. Schematic overview of the assessment points and measures in Gemini that are used in this thesis

Assessment	Twin Age Date Response rate, n families [% of baseline]	**0-1 years** 2007-2008 2402 (100%)	**1-2 years** 2008-2009 1930 (80%)	**2-3 years** 2009-2010 1364 (57%)	**3-4 years** 2010-2012 1119 (47%)	**5-6 years** 2012-2013 1087 (45%)
Socio-demographics	Maternal education	x				
	Maternal ethnicity	x				
Twin characteristics	Twin date of birth	x				
	Twin birth weight	x				
	Anthropometrics	x		x	x	x
Home environment	Siblings in household		x		x	
	Home food environment				x	
	TV viewing		x		x	
	CHAOS					x
Eating behaviour	Appetite - CEBQ		x			x
	Daily energy intake averaged from 3-day diet diaries			x		
Sleep behaviour	Bedtime and wake time		x			x

4.3.3 Sleep

Nighttime sleep duration has been assessed at multiple time points in the Gemini study (see Table 4.3). Sleep was first assessed when the children were on average 16 months old (mean 15.7, SD 1.1 months). At this age, the primary caregiver reported on several aspects of sleep behaviour including daytime sleep, whether their child regularly woke up at night (yes/no), as well as their child's normal bedtime and wake time.

Sleep was assessed again when the children were 5 years old (mean 5.15, SD 0.13 years) using the Children's Sleep Habits Questionnaire (Owens, Spirito, & McGuinn, 2000). This is a validated parent-report questionnaire designed to assess a number of key domains of sleep behaviour in children aged 4-10 years. These include: bedtime behavior and sleep onset; sleep duration; anxiety around sleep; behavior occurring during sleep and night wakings; sleep-disordered breathing; parasomnias; and morning waking/daytime sleepiness (Owens, et al., 2000). This measure was recommended as the best available questionnaire for assessment of childhood sleep behaviour in discussion with a sleep expert (Dr Alice Gregory, Goldsmiths University).

Although multiple aspects of sleep were assessed at 16 months and 5 years, nighttime sleep duration was the primary end-point of interest in this thesis. This was deemed appropriate as it would aid comparison with previous work in the field, the majority of which has examined the relationship between sleep duration and adiposity (this literature was reviewed in Chapter 2, section 2.1). Furthermore, there is evidence that parents are less able to accurately report other aspects of child sleep, such as night waking (Kushnir et al., 2013). Therefore, investigating dimensions of nighttime sleep beyond duration may be better achieved in studies utilising objective measures of sleep behaviour, which was not feasible in the Gemini sample.

At both 16 months and 5 years nighttime sleep duration was calculated from parent reported bedtime and wake time. Calculating sleep duration in this way is common in large population-based studies, and has been validated against actigraphy in young children (Sadeh, Raviv, & Gruber, 2000; Avi Sadeh, 2004). For example, a recent validation study in a sample of preschool-aged children found a strong correlation ($r = 0.85$) between parent-reported nighttime sleep and actigraphy measured sleep duration (Kushnir et al., 2013), although it is understood that parent-report may be prone to overestimate nighttime sleep duration by approximately 30 minutes (Nelson et al., 2014). However, given night to night variability in sleep duration, parent-report may also provide a better global representation of sleep duration than a few nights of objective recording (Sadeh, 2004). Encouragingly, in a subsample of 40 families one week test-retest reliability of the sleep items was high (intraclass correlation 0.89; 95% CI 0.76 – 0.95 for nighttime sleep duration). Descriptive information for nighttime sleep measured at each time point in Gemini is shown in Table 4.1. There was moderate correspondence between nighttime sleep duration measured at 15 months and 5 years ($r = 0.233$, $P < 0.001$). Short sleepers at 15 months were 2.8 times more likely to be short sleepers at age 5 years (OR 2.8, 95% CI 1.77 to 4.63, $P < 0.001$). A copy of all sleep questionnaires can be found in Appendix B.

Table 4.4. Descriptive information for nighttime sleep duration measured at each time point in Gemini

	Total n	Mean (hours)	SD
15 months	1925	11.60	0.86
5 years	1040	11.52	0.60

4.3.4 Dietary intake

The Gemini dietary data is the largest dietary data set of its kind for toddlers in the UK. Dietary intake was measured using 3-day diaries, completed by the primary caregiver when the children were on average 21 months old (mean 20.7, SD 1.6). Parents were sent the diaries alongside detailed instructions and were asked to record everything their child ate or drank inside or outside the home for 3 days, including two weekdays and one weekend day while in their care. Each dietary day covered a 24-hour period, and for each entry, parents recorded the time of the eating occasion. An example diary for one day is shown in Appendix C.

Detailed instructions were provided to parents and caregivers on how to estimate the portion size of all consumed food and drinks. In the first instance, parents were asked to use household measures to estimate the food eaten. In the absence of household measures, they were asked to use packet weights, and in the absence of both, they were asked to estimate the portion size using a booklet of photographs provided with the diaries (the booklet is shown in Appendix C). This booklet illustrated a wide range of example portion sizes, and parents were advised that the photos could be used for foods not shown (e.g. pasta looks similar to rice; shepherd's pie looks similar to lasagna).

Nutrition data from the diaries were coded by researchers at the Medical Research Council Human Nutrition Research unit (MRC HNR) at the University of Cambridge. Energy intakes were estimated by matching each food reported in the diary to a British food composition database (Foster, Hawkins, Adamson, & Food Standards Agency, 2010) using 'Diet In Nutrients Out' (DINO), an in-house program developed by the MRC HNR (Food Standards Agency, 2002), then multiplying by the portion size estimate provided by the parent.

When coding the diaries, all food and drink items within a single time entry (a single clock-time) constituted an eating occasion. Each occasion was individually coded as a meal or snack using a food-based classification system (Macdiarmid et al., 2009). If an eating occasion included a 'meal' food item (e.g. meat, fish, eggs, cooked vegetables), it was coded as a meal, and if it consisted of one or more snack items (e.g. a cookie, crisps, yogurt, raw vegetables) without a meal item, it was coded as a snack (the coding frame is provided in Appendix C). A drink occasion was defined as a drink without any foods, and included milk drinks and water. This coding frame ensured meal, snack and drink occasions were consistently defined. Parents also provided information on whether each occasion was a meal, snack or drink, and the agreement rate between the coding frame and parents' classification was high ($\kappa = 0.82$, $P < 0.001$). However, the parent-derived classifications were not used because a large proportion of these data were missing (approximately 20%), it was therefore agreed that a food-based classification system could provide a more consistent definition of a meal, snack or drink occasion.

To assist in coding missing data, general dietary questions were included with the diaries. For example, one question asked 'What type of milk do your twins most often drink', with separate response options for formula milk, cow's milk whole, cow's milk semi-skimmed, cow's milk skimmed, or other (with instructions to specify type and brand). In the absence of enough detail in the diary, the coders would refer to these general questions. In the absence of both detail in the diary and a response to the general questionnaire, an assumption would have been made. For example, regarding milk consumption, the standard assumption would be based on the type of milk that is most commonly consumed among children of this age, which at the time of dietary assessment was whole cow's milk. The general questions included alongside the diet diaries are given in Appendix C.

The method of dietary data collection used in the Gemini study outperforms alternative unweighed methods, and has been validated against weighed dietary records in children aged 6 to 24 months (Lanigan, Wells, Lawson, & Lucas, 2001). However, the accuracy of any paediatric dietary data is ultimately determined by the parent's ability to correctly recall food consumed and portion size. While the portion size booklets were provided to help standardise the reporting procedure, there will of course be some degree of variability. Furthermore, completing a 3-day diet diary is a time-consuming task and this may have created a bias between families who were more, and those who were less, invested in the Gemini study. Nonetheless, it is encouraging that mean daily energy intake reported in the Gemini diaries is comparable to energy intake data for children aged 12-18 months reported in the Diet and Nutrition Survey of Infants and Young Children in 2011 (967 kcal/day) (Lennox, Sommerville, Ong, Henderson, & Allen, 2011). Mean reported intakes were also comparable to dietary references values for children of this age. Table 4.5 shows the mean intake of energy (kcal/day) and macronutrients from both food and beverages for all children with dietary data, and comparison of these values with dietary reference values. An example copy of a diet diary, portion size booklet, dietary questions, and coding frame can be found in Appendix C.

4.3.4.1 Underreporting

Dietary data is prone to reporting error (Livingstone, Robson, & Wallace, 2004). Table 4.5 shows 37% of the Gemini sample report energy intakes that do not meet Daily Recommended Values (DRV), while less than 10% of the sample are reported as ‘thin’ (Table 4.8), which may indicate underreporting in the data. In order to assess the degree of reporting error in the Gemini dietary data, analyses were run comparing estimated energy requirements (EER) to reported daily energy intake. The basic assumption for assessing underreporting is that where reported energy intake does not equal EER there is evidence for reporting error. However, an upper and lower plausible level of both energy intake and EER are also calculated in order to allow for inherent variation in the estimation of both factors. This is achieved by deriving a ‘coefficient of variation’ (CV_t). The steps below describe the process for estimating EER as well as the CV_t.

1.) EER is calculated from EE and the energy required for growth (Eg): **EER = EE + Eg**

a. **EE** is calculated from body weight (in this case the interpolated weights at diary completion, approximately 21 months) using standard equations, below (equations taken from Torun et al., 2005)

- EE(kcals / day) = 310.2 + 63.3kg – 0.263kg^2 (boys)
- EE(kcals / day) = 263.4 + 65.3kg – 0.454kg^2 (girls)

b. **Eg** = mean weight gain (g/day) * 8.6 (KJ/g)

- Given the age range of sample, weight gain/day used 1-1.9 years (= 6.6g/day for boys and girls)

2.) The coefficient of variation (CVt) of EI/EER is calculated with equation:

$$CV_t = \sqrt{CV_{EE}^2 + \frac{CV_{EI}^2}{d}} = 21\%$$

- d = number of diary days (d=3)
- CV^2_{EE} = CV for measurement of EE (= 19.1%, the average from dietary studies which EER equations are based, Torun, 2005)
- CV^2_{EI} = CV for measurement of EI (= 5.5% at age 21 months. This was calculated from Gemini data by dividing the mean EI for each child by the standard deviations for each child)

3.) Solving the equation, $CV_t = 19.37\%$

Based on the above calculations, energy intake reported between 80.63% and 119.37% (100% +/- 19.37%) of EER was considered within the range of normal measurement error. Children with reported energy intake in this range were classified as 'plausible reporters', those with EI below 80.63% of EER were classified as 'under-reporters', and those with energy intake above 119.37% of EER were identified as 'over-reporters'. Running these analyses on Gemini dietary data revealed that in total 11.9% of all children with dietary data were classified as over-reporters, while 12.3% were under-reporters. Importantly, this indicates that the majority of the sample (73.7%) were plausible reporters. Nevertheless, these results indicate that there is some level of reporting bias in the Gemini dietary data, which should be considered when interpreting the findings presented in this thesis.

Table 4.5. Mean intake of energy (kcal/day) and macronutrients from food and beverages for children (n=2336) aged 21 months in the Gemini sample, and comparison with Dietary Reference Values (DRV)[7]

Nutrient	DRV	Gemini mean[a] (% of DRV)	% of Gemini sample not meeting DRV	SD	25th percentile	75th percentile
Daily energy intake (kcals)	968[b]	1035 (107)[b]	37	187	908	1144
Total fat (g/d)	-	42	-	10	35	49
Total fat (%E)	33[c]	37 (112)	21.5	5	34	40
Saturated fat (g/d)	-	20	-	6	16	24
Saturated fat (%E)	10[c]	18 (180)	1.9	3	15	20
Protein (g/d)	14.5[d]	40 (276)	0.1	9	34	45
Protein (%E)	-	12	-	2	11	14
Total carbohydrates (g/d)	-	132	-	27	114	148
Total carbohydrates (%E)	47[c]	51 (109)	23.9	6	47	55

[a] Mean intake including supplements
[b] DRV for daily energy intake is based on the Scientific Advisory Committee on Nutrition (2011) estimated average requirements (EARs) for children 2 years of age and the mid-point of DRV for males (1004 kcal/d) and females (932 kcal/d)
[c] DRV for children 1-3 years of age from Department of Health, *Dietary Reference Values for Food Energy and Nutrients for the United Kingdom*, HMSO, 1991
d DRV from Scientific Advisory Committee on Nutrition (2003)

[7] Data from this table were adapted with permission from a Gemini publication describing the energy and nutrient intakes of young children in the UK (Syrad et al., 2015). It provides a useful overview of the energy and macronutrient intakes reported by all families who completed the diaries, in comparison with dietary reference values for children of this age.

4.3.5 Home environment

The home environment was assessed when the children were on average 4 years old (mean 4.2, SD 0.4) using a computer-assisted telephone interview completed by trained researchers. Whilst I was instrumental in the collection of the home environment data, and had input on the piloting and design of the survey, the development of the home environment interview and scoring procedure formed a core part of the thesis work of Dr Stephanie Schrempft (a member of the Gemini research team). Therefore, only aspects of the home environment relevant to the current thesis are briefly described.

Multiple constructs of the home food environment were assessed in the Home Environment Interview (HEI). The interview procedure involved a telephone call assisted by an online script. The researchers could enter the caregiver's response directly into the online form, thereby minimising the amount of missing data. The interview was adapted from the Healthy Homes Survey (Bryant et al., 2008) to include measures of parental support for physical activity, parental TV viewing, and neighbourhood satisfaction (Schrempft, van Jaarsveld, Fisher, & Wardle, 2015). This ensured the home environment interview provided a comprehensive assessment of both physical and social aspects of the home food, activity, and media environment. The present thesis used data from the composite measure of the home food environment only; this composite is described below. A copy of the interview transcript can be found in Appendix D.

4.3.5.1 Home food environment composite: development, scoring, and associations with energy balance behaviours

Variables hypothesised to be theoretically relevant to weight gain, or those with empirically established associations with weight were assessed and included in the composite score. These variables were initially identified through a review of the literature, and then verified by a panel of 30 experts in childhood obesity. The experts were sent an email link to an online survey, where they were asked to specify whether a given home environment variable was (or was not) associated with either an increased or decreased risk of weight gain. A given variable was included in the composite score if more than 60% of experts believed it to be associated either an increased or decreased risk of weight gain. In total, data from 21 variables were combined to create the composite risk score for the food environment. Broadly, variables related to food availability, visual accessibility of food, physical accessibility of food, and parental feeding practices. Food availability was assessed in terms of presence (e.g. ‘do you have any fresh fruit in your home now?’) and variety (e.g. ‘what types of fresh fruit do you have in your home now?’). Visual accessibility was assessed by asking parents, for example, ‘without opening any fridge or cupboard doors, is there any kind of fruit in your home now; displayed out in the open?’, and physical accessibility by asking parents, for example, ‘would it be possible for your twins to get any confectionery by themselves, without your help?’. Measures of parental feeding were taken from standardised questionnaires. Specifically, ‘emotional feeding’, ‘instrumental feeding’, ‘encouragement’, and ‘restriction’ were measured using the Parental Feeding Styles Questionnaire (Wardle et al., 2002). ‘Modelling’ was measured using a scale from the Comprehensive Feeding Practices Questionnaire (Musher-eizenman & Holub, 2007), ‘monitoring’ from the Child Feeding Questionnaire (Birch et al., 2001), and ‘covert restriction’ was assessed using an adapted scale developed by Ogden et al. (2006). All parental feeding items were scored on a 5-point scale (1 = never; 5 = always), except for restriction, which was measured on a 7-point scale (1 = not at all; 7 = strictly). A mean score was calculated for each scale, with higher scores indicating higher levels of the particular feeding practice. Table 4.6 shows the variables included in the home food environment composite, along with descriptive statistics.

To create the composite risk score, variables identified as being associated with a decreased risk of weight gain were first reverse scored. Variables were then standardised using z-scores, and missing values were recoded to 0, which is the mean score for a standardised variable. However, as the assessment was administered as a telephone interview, there were very few missing values. Values were then summed to create the composite score, such that a higher score reflected a higher risk home food environment (for weight gain). In a random sample of 40 families, one-week test-retest of the home food environment composite was high (0.71; 95% CI 0.52 – 0.83) (Schrempft et al., 2015).

Table 4.6. Descriptive statistics for the home food environment variables (n=1096 families) Data are % (n) who responded yes unless stated otherwise[8]

Home food environment	
Availability	
Number of fruit types, mean (SD)[a]	7.76 (3.20)
Number of vegetable types, mean (SD)[a]	10.78 (3.74)
Number of energy-dense snack types, mean (SD)	5.22 (2.08)
Presence of sugar-sweetened drinks	38.6 (423)
Accessibility (visibility)	
Fruit on display [a]	93.5 (1025)
Vegetables ready-to-eat [a]	54.0 (592)
Energy-dense snacks on display	20.5 (225)
Sugar-sweetened drinks on display	6.6 (72)
Accessibility (child can help him/herself)	
Fruit [a]	53.4 (585)

[8] Information from this table was adapted with permission from the Gemini development paper for the home environment interview, Schrempft et al. (2015).

Vegetables [a]	28.3 (310)
Energy-dense snacks	8.7 (95)
Sugar-sweetened drinks	2.0 (22)
Parental feeding practices, mean (SD)	
Emotional feeding [b] (1 = never; 5 = always)	1.80 (0.62)
Instrumental feeding [b] (1 = never; 5 = always)	2.18 (0.66)
Encouragement [a, b] (1 = never; 5 = always)	4.12 (0.54)
Modelling [a, b] (1 = never; 5 = always)	3.63 (0.75)
Monitoring [a, c] (1 = never; 5 = always)	3.68 (0.91)
Covert restriction [a, c] (1 = never; 5 = always)	3.02 (0.83)
Restriction [a, d] (1 = not at all; 7 = strictly)	5.19 (1.11)
Family meal frequency (days per week)	3.83 (1.62)
Frequency child eats while watching TV (days per week)	1.32 (1.52)

a - Variable was identified as being associated with decreased risk for weight gain

b - Variable had 39 missing values

c - Variable had 40 missing values

d - Variable had 42 missing values

Previous work has shown that Gemini children living in higher risk home food environments are more likely to consume energy-dense snacks and sugar sweetened beverages, and less likely to consume fruits and vegetables (Schremfpt et al., 2015). Despite being associated with dietary intake, the home food environment risk score was not associated with BMI-SDS at age 4 years (Schremfpt et al., 2015). It may be that home-environmental influences on weight emerge later in childhood, and strengthen overtime. Research in older samples of children (aged 10-12 years) for example has shown that composite measures of the home environment are associated with BMI (MacFarlane, Cleland, Crawford, Campbell, & Timperio, 2009).

4.3.6 Chaos

Chaos in the home environment was measured when the twins were on average 5 years old (mean 5.15, SD 0.13 years) using the Confusion Hubbub and Order (CHAOS) Scale (Matheny, Wachs, Ludwig, & Phillips, 1995). The CHAOS scale is a forced choice questionnaire designed to assess the general level of confusion, disorganisation and noise within the home (for example, 'you can't hear yourself think in our home'). Items are responded to as true (1 point) or false (2 points) and scores are averaged, such that higher scores indicate a greater level of chaos within the home. Mean scores were calculated if at least 4 out of a possible 6 items were completed. A copy of the CHAOS items can be found in Appendix E.

The CHAOS scale has been shown to correlate well with objective measures of home disorganisation and parenting, and shows good internal consistency (Cronbach's α= .79) and 12-month stability (r = 0.74) (Matheny et al., 1995). Importantly, previous work has shown that home chaos is not a proxy for adverse social circumstances or socioeconomic status, but is a meaningful construct in its own right (Dumas et al., 2010). For example, a greater level of home chaos has been associated with poorer cognitive, behavioural, and self-regulatory outcomes in young children, independent of potential confounding factors such as socioeconomic status (Dumas et al., 2010). Importantly, a greater degree of chaos in the home has also been associated with higher food responsiveness, shorter sleep duration, as well as adiposity in paediatric samples (Appelhans et al., 2014; Lumeng et al., 2014).

4.3.7 Eating behaviour phenotypes

Eating behaviour was measured using the Children's Eating Behaviour Questionnaire (CEBQ) when the twins were around 5 years old (mean 5.15, SD 0.13 years) (Wardle, Guthrie, Sanderson, & Rapoport, 2001). The CEBQ is a 35-item instrument that measures 7 eating behaviours, and a drinking behaviour implicated in the development of paediatric obestity (Wardle et al., 2001). The constructs measured and included within this thesis were 'food responsiveness' and 'satiety responsiveness'. Parents are asked to reference their child's habitual eating style, and responses to each item were provided on a Likert scale from 1 (never) to 5 (always). Mean scores for each subscale were calculated if at least 3 out of a possible 5 items were completed. The food responsiveness scale has 5 items assessing the degree to which a child expresses a desire for food, particularly in response to palatable foods (e.g. 'my child is always asking for food'). The satiety responsiveness scale has 5 items assessing the degree to which a child tends to stop eating (or doesn't initiate eating) according to their perceived fullness (e.g. 'my child cannot eat a meal if s/he has had a snack just before'). A copy of the items included in each scale can be found in Appendix F.

Both food responsiveness and satiety responsiveness have been shown to have good test-retest reliability ($r = .85$ and $r = .83$, respectively), high internal consistency (Cronbach's $\alpha = .83$ and $\alpha = .82$, respectively), and have been validated against behavioural measures of food intake in children (Wardle et al., 2001). For example, in a behavioural validation task in 4-5 year old children (completing a battery of eating behaviour tasks), higher satiety responsiveness was associated better caloric compensation after preload, a slower rate of eating, and a lower average energy intake. Conversely, higher food responsiveness was associated with a faster rate of eating and a higher average energy intake (Carnell & Wardle, 2007). Indeed, both lower satiety responsiveness and higher food responsiveness reflect behavioural phenotypes associated with obesity risk, and there is now a large body of research demonstrating that children with lower satiety responsiveness and higher food responsiveness tend to be heavier, and gain more weight over time (e.g. van Jaarsveld, Boniface, Llewellyn, & Wardle, 2014; Viana, Sinde, & Saxton, 2008; Webber et al., 2009). Table 4.7 shows descriptive and scale information for food responsiveness and satiety responsiveness measured at age 5 years.

Table 4.7. Descriptive and scale information for food responsiveness and satiety responsiveness measured in Gemini at age 5 years

Trait	Number of items	Example item	Cronbach's alpha	Mean (SD)	Range
Food responsiveness[1]	5	My child is always asking for food	0.81	2.37 (0.75)	1.0 to 5.0
Satiety Responsiveness[2]	5	My child cannot eat a meal if s/he has had a snack just before	0.75	2.84 (0.62)	1.0 to 5.0

[1]n = 1052, [2]n = 1049

4.3.8 Anthropometrics

At baseline, parents were asked to provide the length, head circumferences, and weight of both twins at birth by copying or transcribing their twins' health records. When the twins were 2 years old, parents were sent electronic weighing scales (Tanita UK, Yewsley, UK), and height charts, and instructions to provide on-going 3-monthy measurements (with replacements provided upon request). Reminders were sent every 3 months by email and/or mail, and parents could submit measurements using an online form, by email, telephone or post. Importantly, there is evidence to show that parents can accurately weigh and measure their twins at home, when provided with standardised equipment (Himes, 2009). BMI was then calculated from children's height and weight using the equation weight (kg) / height (m)^2, and was transformed into age- and sex- specific BMI-SDS (according UK 1990 reference data) using the Imsgrowth macro (http://homepage.mac.com/tjcole). The height and weight data included in this thesis were extensively cleaned by our data manager (an epidemiologist), with growth trajectories examined for each child. Table 4.8 shows descriptive information for all BMI data available between 24 and 63 months of age according to the International Obesity Taskforce (IOFT) classification for adiposity (Cole, Bellizzi, Flegal, & Dietz, 2000). The international cut-offs are provided as they classify BMI in children aged 2-18 years as thin, normal weight, overweight or obese, depending on the child's age and sex, based on adult BMI cut-offs at 18 years. The cut offs and corresponding grades are also shown in Table 4.8.

Table 4.8. BMI IOTF classification at 3 month intervals for all available data from age 2 years onwards

Age	Thinness grade 3 n (%)	Thinness grade 2 n (%)	Thinness grade 1 n (%)	Normal weight n (%)	Over-weight n (%)	Obese n (%)	Total n
BMI range at 18 y	<16	16 to <17	17 to <18.5	18.5 to <25	25 to <30	30+	
24 months	17 (1.2)	14 (1.0)	94 (6.6)	1057 (74.1)	201 (14.1)	43 (3.0)	1426
27 months	4 (0.3)	26 (1.9)	87 (6.4)	1047 (76.5)	173 (12.6)	31 (2.3)	1368
30 months	4 (0.3)	26 (2.2)	81 (6.8)	907 (75.8)	144 (12.0)	34 (2.8)	1196
33 months	5 (0.4)	17 (1.5)	103 (8.9)	898 (77.8)	108 (9.4)	23 (2.0)	1154
36 months	17 (1.3)	23 (1.7)	124 (9.3)	1021 (76.3)	124 (9.3)	29 (2.2)	1338
39 months	11 (0.8)	24 (1.8)	124 (9.4)	1009 (76.7)	126 (9.6)	22 (1.7)	1316
42 months	9 (0.8)	20 (1.7)	111 (9.3)	917 (76.5)	121 (2.5)	20 (1.7)	1198
45 months	6 (0.6)	11 (1.2)	84 (8.9)	742 (78.4)	93 (9.8)	10 (1.1)	946
48 months	7 (0.8)	16 (1.9)	70 (8.2)	680 (79.3)	75 (8.7)	10 (1.2)	858
51 months	7 (1.0)	12 (1.7)	60 (8.5)	573 (81.3)	48 (6.8)	5 (0.7)	705
54 months	2 (0.3)	9 (1.4)	65 (10.2)	512 (80.0)	47 (7.3)	5 (0.8)	640
57 months	7 (1.1)	16 (2.4)	71 (10.8)	518 (78.8)	37 (5.6)	8 (1.2)	657
60 months	12 (1.5)	12 (1.5)	92 (11.3)	640 (78.9)	46 (5.7)	9 (1.1)	811
63 months	11 (2.2)	9 (1.8)	68 (13.5)	376 (74.8)	35 (7.0)	4 (0.8)	503

4.3.9 Twin characteristics

The twins' date of birth, sex, and gestational age were reported in the baseline questionnaires. Age at each follow-up questionnaire was calculated using the twins' date of birth and the date of questionnaire completion.

4.3.10 Sociodemographic data: maternal education and ethnicity

Maternal education and ethnicity were reported in the baseline questionnaires. The primary caregiver reported the mother's highest education level, which was categorized as: lower (compulsory schooling only), middle (some additional school or vocational examinations), and higher (university education). Throughout this thesis, maternal education was used as a proxy measure for socioeconomic status. Because socioeconomic status is understood to have a widespread influence on health outcomes, including sleep duration and obesity, this factor was controlled for in in each of the study chapters. Although other measures are available to index socioeconomic status, a recent systematic review demonstrated that, of all indicators of socioeconomic status, maternal education is one of the most consistent predictors of childhood adiposity (Shrewsbury & Wardle, 2008).

In the baseline questionnaires families were also asked to report the mother's ethnicity using recommended classifications from the Office for National Statistics, which included: white British; white Irish; other white background; Caribbean; African; other black background; Indian; Pakistani; Bangladeshi; other Asian background; white and black Caribbean; white and black African; white and Asian; other mixed background; Chinese; any other. The categories were combined into 'white' and 'non-white' as there were too few non-white participants to analyse the different groups.

4.4 Summary and comment

The Gemini study is a population-based cohort of young children that has included multiple assessments of nighttime sleep duration, height and weight, and energy-balance behaviours (in particular eating behaviour and dietary intake). The majority of data has been collected from paper-based or online questionnaires completed by the primary caregiver, predominantly the mother. Although collecting objective measures of sleep duration and adiposity alongside these measures would have made a valuable addition, the resource costs associated with doing so meant this was not feasible. The questionnaire data used in this thesis is however of sound methodological quality, and the dietary data is the largest of its kind for toddlers in the UK. While the baseline Gemini sample reflects a representative population-based cohort, there has been some degree of attrition over the 6-year study period. The strengths and limitations relevant to data collection and the study population as a whole are addressed in detail in the General Discussion.

Chapter 5 Study 1: Predictors of shorter sleep in early childhood[9]

5.1 Background and aims

As outlined in Chapter 2 (section 2.1) insufficient sleep in early childhood is increasingly being shown to be associated with adverse health outcomes; notably an increased risk of obesity (Chen et al., 2008). The health implications of short sleep make it important to understand its determinants, particularly in early childhood (around 15-18 months of age), during which twin studies suggest there may be a critical window for environmental influence (Touchette et al., 2013), and when patterns of sleep behaviour may be being established (Byars, Yolton, Rausch, Lanphear, & Beebe, 2012). For example, in Gemini, short sleepers at 15 months were 2.8 times more likely to be short sleepers at age 5 years (OR 2.8, 95% CI 1.77 to 4.63, $p < 0.001$).

As reviewed in Chapter 1 (section 1.3.4), multiple features within a child's physical and social environment have been associated with shorter nighttime sleep. In particular, there are marked ethnic and socioeconomic disparities in sleep behaviour with children from ethnic minority groups and more poorly educated backgrounds significantly more likely to experience short sleep at night. Certain exposures within the environment, notably increased screen time and a greater degree of noise or chaos have also been shown to be detrimental (Chapter 1, section 1.3.4). Although multiple features within the physical and social home environment can influence sleep in early life, it is not known whether these effects are mediated by the timing of bedtime or wake time. This information could help to understand the mechanisms of shorter sleep and inform strategies to improve sleep in early life.

[9] A version of this chapter has been published as: McDonald, L., Wardle, J., Llewellyn, C. H., van Jaarsveld, C. H. M., & Fisher, A. (2014). Predictors of Shorter Sleep in Early Childhood. *Sleep Medicine*, *15*, 536-540.

Therefore, the aim of this study was to identify predictors of shorter sleep at night in children aged 16 months, and to test whether predictors operated primarily through sleep initiation (bedtime) or sleep termination (wake time).

5.2 Participants and methods

Participants were 1,702 children from the Gemini twin birth cohort, which is described in detail in Chapter 4. For the present study, one child from each family was randomly selected to avoid non-independence of data. There were no differences between mothers who provided complete data and those who did not for age (33.3 versus 33.5 years at twin birth, P = 0.30), BMI (24.9 versus 25.0 at baseline, P = 0.20), or education level (P = 0.09). However, slightly more mothers who provided complete information were from a white British ethnic background (88.7% versus 83.9%, P = 0.02).

5.2.1 Sleep

As described in Chapter 4 (section 4.3.3), sleep was assessed when the children were on average 16 months old (mean 15.7, SD 1.1 months), and nighttime sleep duration was calculated from parent-reported bedtime and wake time.

There are currently no clinical recommendations to define short sleep in early childhood, but population data can provide an indication of basic sleep requirements (Iglowstein et al., 2003). At around 18 months of age, population means from a sample of 11,000 British children suggest children of this age sleep on average 11.3 (SD 1.3) hours per night (Figure 1.3; Blair et al., 2012), although individual variability exists. Sleeping <11 hours per night at this age is also associated with an increased risk of obesity (Bell & Zimmerman, 2010; Chen et al., 2008), so may reflect a level of sleep that is not sufficient to support optimal functioning or health. Therefore, shorter sleep in this study was defined as sleep <11 hours per night (Chen et al., 2008). A sensitivity analysis was also conducted using a cut point at the 10^{th} percentile of sleep duration to define 'short' sleep (<10.5 hours per night). Patterns of results were the same, so data are presented using <11 hours as the demarcation for short sleep. The results of the sensitivity analysis are shown in Appendix G.

5.2.2 Socio-demographic and environmental characteristics

Socio-demographic risk factors included maternal education (up to secondary school, college and beyond), maternal ethnicity (white, non-white) and child sex (male, female). Birth weight was categorised as low (≤2500 grams) or normal weight (>2500 grams) (Kramer, 1987). Environmental factors reported when the children were 16 months old included the number of older children living in the home (0, 1, >1) and hours of TV viewing in the morning (up to midday), and after 6:30 in the evening (≤1, >1 hour).

5.2.3 Statistical analyses

Logistic regression models were used to identify significant predictors of shorter sleep. Models were first run separately for each factor, followed by a multiple logistic regression model. All models were adjusted for age because the range in the sample (14 to 27 months) was too narrow to study age effects, and for daytime sleep because at this age daytime sleep still represents a large proportion of total sleep duration (Iglowstein et al., 2003) and shows considerable ethnic variation (Crosby, LeBourgeois, & Harsh, 2005). All models were also adjusted for regular night waking, as this may disrupt the normal sleep-wake cycle.

To test whether the predictors of shorter nighttime sleep operated primarily through wake time or bedtime, a standard, four-step mediation approach was used (Baron & Kenny, 1986). Mediation is considered to take place when (1) the mediator (wake time or bedtime) significantly predicts the dependent variable (shorter sleep), (2) the independent variable significantly predicts the dependent variable, (3) the independent variable significantly predicts the mediator, and (4) the association between the independent variable and the dependent variable is substantially reduced when the mediator is included in the model. Criteria 1 and 2 were assessed with the logistic regression and multiple logistic regression models used to identify predictors of shorter sleep. Criterion 3 was assessed using two separate multiple linear regression models predicting wake time and bedtime. Criterion 4 was assessed using two separate multiple logistic regression models including all determinants, and either wake time or bedtime. Where the results satisfied criteria for mediation, the Sobel test was used as the test of significance (http://www.psych.ku/preacher/sobel).

5.3 Results

Complete data were available for 1,702 children (71.0% of baseline sample). Sleep duration was normally distributed. The average nighttime sleep duration in the sample was 11.6 hours per night (SD 52 minutes) and the average daytime sleep was 1.9 hours (SD 41 m). Shorter sleep (<11 hours per night) was reported in 14.1% of the sample. There was no significant association between daytime and nighttime sleep duration, indicating that shorter nighttime sleepers were not compensating during the day. Table 5.1 shows the percentage of children experiencing shorter nighttime sleep by family characteristics. Shorter nighttime sleep was more common in children from ethnic minority families and those where the mother had less education. Shorter sleep was also more common in boys, and children watching more than 1 hour of TV in either the morning or evening.

The average bedtime of the shorter-sleeping group was 8:05pm (SD 61m), compared with 7:04pm (SD 33m) in those with ≥11 hours of sleep. The average wake time of the shorter-sleeping group was 6:14am (SD 54m) compared with 6:56am (SD 39m) in those with ≥11 hours of sleep. There was a small positive correlation between bedtime and wake time, such that those children who went to bed later tended to wake up slightly later as well ($r = 0.29$, $P < 0.001$).

5.3.1 Predictors of Shorter Sleep

Table 5.1 presents the logistic regression models predicting shorter sleep. Lower maternal education (odds ratio [OR]= 1.64, 95% confidence interval: 1.23 to 2.17), coming from a minority ethnic background (OR = 5.10, 95% CI: 3.16 to 8.24), being male (OR = 1.45, 95% CI: 1.08 to 1.92), and having been born at a low birth weight (OR = 1.43, 95% CI: 1.07 to 1.90) significantly increased the odds of shorter sleep. Having more than one older child in the home (OR = 1.70, 95% CI: 1.17 to 2.47) and watching more than an hour of TV in the morning (OR = 1.47, 95% CI 1.10 to 1.96) or evening (OR = 2.22, 95% CI: 1.55 to 3.18) were also associated with shorter sleep. In the multiple logistic regression analysis with all variables included in the model, the effect of morning TV was no longer significant, but all other effects remained significant (Model 2 in Table 5.1).

5.3.2 Pathways to Shorter Sleep

As expected, because nighttime sleep is a function of both bedtime and wake time, children who woke up later in the morning (OR = 0.22, 95% CI: 0.17 to 0.29) were less likely to experience shorter sleep, and those who went to bed later in the evening were substantially more likely to be shorter sleepers (OR = 6.04, 95% CI: 4.72 to 7.72). Table 5.2 shows associations with wake time and bedtime for all variables that independently predicted shorter sleep. The only predictor of earlier waking was being male; other effects were either non-significant or associated with *later* wake times. In contrast, lower maternal education, ethnic minority status and watching >1 hour of evening TV were associated with a later bedtime.

The results of adding either wake time (Model 1) or bedtime (Model 2) into the multiple logistic regression model predicting shorter sleep are shown in Table 5.3. These can be compared with the odds ratios shown in Table 5.1 (Model 2). After including wake time, all determinants remained significant predictors of shorter sleep with the exception of sex. The Sobel test showed significant mediation of the gender effect by wake time ($P < 0.001$). Other determinants did not satisfy the criteria for mediation by wake time.

Including bedtime in the model reduced the odds ratios associated with maternal education, ethnicity, and time spent watching TV in the evening to non-significance. Sobel tests indicated a later bedtime significantly mediated the associations between lower education ($P < 0.001$), ethnic minority status ($P < 0.001$), and evening TV viewing ($P < 0.001$) and shorter sleep. Other determinants did not satisfy the criteria for mediation by bedtime.

Table 5.1. Percentage of participants reporting shorter sleep by family characteristics and logistic regression models predicting shorter night time sleep

Risk Factors	**Shorter Sleep (<11 hours)** **n (%)**	**Model 1 (Simple logistic regression)** OR (95% CI)	**Model 2 (Multiple logistic regression)** OR (95% CI)
Total (n = 1702)	240 (14.1)		
Maternal education			
High (n=964)	108 (11.2)	1.00	1.00
Low (n=738)	132 (17.9)	1.64 (1.23 to 2.17)**	1.46 (1.07 to 1.99)*
Ethnicity			
White (n=1615)	200 (12.4)	1.00	1.00
Non-White (n=87)	40 (46.0)	5.10 (3.16 to 8.24)**	5.05 (3.08 to 8.27)**
Sex			
Female (n=874)	104 (11.9)	1.00	1.00
Male (n=828)	136 (16.4)	1.45 (1.08 to 1.92)*	1.61 (1.19 to 2.17)*
Birth weight (grams)			
> 2500 (n=858)	105 (12.2)	1.00	1.00
≤ 2500 (n=844)	135 (16.0)	1.43 (1.07 to 1.90)*	1.45 (1.07 to 1.96)*
Birth weight z-score		0.84 (0.72 to 0.79)*	0.85 (0.73 to 0.99)*
Older children			
0 (n=889)	121 (13.6)	1.00	1.00
1 (n=563)	66 (11.7)	0.84 (0.61 to 1.17)	0.83 (0.58 to 1.17)
>1 (n=250)	53 (21.2)	1.70 (1.17 to 2.47)*	1.58 (1.06 to 2.35)*
Morning TV (hours)			
≤1 (n=1176)	141 (12.0)	1.00	1.00
>1 (n=526)	99 (18.8)	1.47 (1.10 to 1.96)*	1.13 (0.80 to 1.58)
Evening TV (hours)			
≤1 (n=1489)	183 (12.3)	1.00	1.00
>1 (n=213)	57 (26.8)	2.22 (1.55 to 3.18)**	1.89 (1.26 to 2.84)*

(**Table 5.1** *continued*)

Risk Factors	Shorter Sleep (<11 hours) n (%)	Model 1 (Simple logistic regression) OR (95% CI)	Model 2 (Multiple logistic regression) OR (95% CI)
Mediators			
Wake time (per hour)	-	0.22 (0.17 to 0.29)**	-
Bedtime (per hour)	-	6.04 (4.72 to 7.72)**	-

Abbreviations: *P < 0.05; ** P ≤ 0.001; OR, odds ratio; CI, confidence interval
Model 2 is a multiple logistic regression model containing all risk factors predicting shorter sleep. All models adjusted for age, daytime sleep and regular night waking.

Table 5.2. Multiple linear regression models predicting wake time and bedtime

	Wake time		Bedtime	
Risk Factors	**B (SE)**	**P-value**	**B (SE)**	**P-value**
Maternal education				
Low vs. High	0.04 (0.04)	0.226	0.11 (0.03)	0.001
Ethnicity				
Non-White vs. White	0.31 (0.08)	<0.001	0.80 (0.08)	<0.001
Sex				
Male vs. Female	-0.16 (0.03)	<0.001	0.02 (0.03)	0.529
Birth weight (grams)				
≤ 2500 vs. > 2500	0.02 (0.04)	0.643	0.01 (0.03)	0.768
Older Children				
>1 vs. 0 vs. 1	-0.02 (0.02)	0.355	-0.03 (.02)	0.259
Evening TV (hours)				
>1 vs. ≤1	0.17 (0.05)	0.001	0.41 (0.05)	<0.001

Abbreviations: B=unstandardized regression coefficient; SE = standard error. Models adjusted for age, daytime sleep and regular night waking.

Table 5.3. Logistic regression models predicting shorter sleep including either wake time (model 1) or bedtime (model 2)

Risk Factors	**Model 1 (including wake time)** OR (95% CI)	**Model 2 (including bedtime)** OR (95% CI)
Maternal education		
High	1.00	1.00
Low	1.55 (1.10 to 2.17)*	1.16 (0.82 to 1.64)
Ethnicity		
White	1.00	1.00
Non-White	12.63 (6.95 to 22.94)**	1.73 (0.92 to 3.26)
Sex		
Female	1.00	1.00
Male	1.24 (0.89 to 1.72)	1.73 (1.23 to 2.43)*
Birth weight (grams)		
> 2500	1.00	1.00
≤ 2500	1.46 (1.05 to 2.04)*	1.52 (1.08 to 2.14)*
Older Children		
0	1.00	1.00
1	0.79 (0.54 to 1.16)	1.01 (0.67 to 1.48)
>1	1.79 (1.15 to 2.79)*	1.34 (0.83 to 2.16)
Evening TV (hours)		
≤1	1.00	1.00
>1	3.02 (1.97 to 4.64)**	1.06 (0.67 to 1.66)

Abbreviations: *P < 0.05; **P ≤ 0.001; OR, odds ratio; CI, confidence interval
Model 1 is a multiple logistic regression model including all risk factors and wake time predicting shorter sleep. Model 2 is a multiple logistic regression model including all risk factors and bedtime predicting shorter sleep. All models are adjusted for age, daytime sleep and regular night waking.

5.4 Discussion

This study helps to establish predictors of shorter nighttime sleep in early life, and identifies several key influences that operate predominantly through a later bedtime. Understanding the importance of an early and consistent bedtime could help to promote healthy sleep in early life and reduce inequalities in child health.

In this study, ethnicity and maternal education emerged as significant influences on sleep in early childhood. Children living in non-white families or families with lower maternal education were more likely to sleep for <11 hours a night. This finding supports a body of literature citing sociocultural differences in sleep behaviour (Mindell, Sadeh, Kwon, & Goh, 2013; Spilsbury et al., 2004), and emerges probably because both maternal education and ethnicity affect multiple features of the child's physical and social environment, including parental attitudes and practices surrounding sleep behaviour (McLaughlin Crabtree et al., 2005; Milan, Snow, & Belay, 2007; Thompson & Christakis, 2005). Importantly, ethnic differences in sleep were partly explained by later bedtimes in ethnic minority groups. Bedtime practices are often culturally defined (Mindell, Sadeh, Wiegand, et al., 2010), and the present results indicate that they may drive disparities in sleep behaviour. Interestingly, children from ethnic minority backgrounds also reported later morning wake times, suggesting a tendency towards a later sleep schedule or propensity to offset sleep loss at night. Maternal education differences were also driven principally by later bedtimes in the lower education group; with no differences in wake times. While education and ethnicity are established risk factors for shorter sleep at night (Mindell et al., 2013; Nevarez, Rifas-Shiman, Kleinman, Gillman, & Taveras, 2010), they are not themselves modifiable; these groups might need specific emphasis on the importance of an early bedtime in determining sleep in early life.

Children who watched more than an hour of TV in the evening were also substantially more likely to be shorter sleepers. This supports previous research demonstrating that longer periods of TV exposure are predictive of greater bedtime resistance, more difficulty maintaining sleep, and shorter sleep duration (Nevarez et al., 2010; Paavonen, Pennonen, Roine, Valkonen, & Lahikainen, 2006; Thompson & Christakis, 2005). Morning TV viewing did not independently predict shorter sleep. While evening TV viewing may be partly a compensating activity for sleep, it could also impair a child's ability to initiate sleep by raising levels of arousal and light exposure before bedtime. Later bedtimes also accounted for the association between time spent watching TV in the evening and risk of shorter sleep. Children watching more TV in the evening also woke up significantly later in the morning; possibly indicating a tendency to compensate for sleep lost at night, although it could reflect broader family patterns. However, the tendency toward later morning wake times was insufficient to offset sleep lost at night. Children under 2 years are recommended not to watch any TV (Committee on Public Education, 2001), however many parents may incorporate TV viewing into their child's bedtime routine (Milan et al., 2007). Engaging in bedtime practices that do not include TV exposure could help children maintain an early bedtime and encourage healthy sleep during early childhood.

The importance of an early bedtime is emphasised in many 'sleep hygiene' recommendations. They often advocate that children maintain a bedtime before 9pm, a consistent bedtime routine, and sleep independently in a quiet, dark room without the presence of media devices (Mindell et al., 2009). In the present study, even children in the shorter sleeping group went to bed well before 9pm, suggesting these recommendations may need to be amended for young children who require more sleep at night and may not consistently compensate by waking later or sleeping longer during the day.

Although several domains of the environment influence sleep in early life, not all operate in the same way. Boys were more likely to experience shorter sleep than girls, and this seemed to be due largely to earlier wake times in boys. Gender-specific differences in sleep patterns have been reported previously (Blair et al., 2012), with some evidence suggesting males are more likely to sleep for shorter periods at night, and experience a lower percentage of motionless or quiet sleep (Goodlin-Jones, Burnham, Gaylor, 2001; Sadeh et al., 2000). Birth weight and the number of older children living in the home also emerged as independent predictors of shorter sleep, but appeared to exert a more general influence on sleep duration and were not associated with either bedtime or wake time. Living in a home with more people is associated with a greater level of chaos (Dumas et al., 2010), so this could disrupt either end of the nighttime sleep episode.

Importantly, all predictors emerged independently of daytime sleep. Although daytime sleep still represents a large proportion of total sleep time in this age group (Blair et al., 2012), daytime sleep did not differ significantly between shorter and longer sleepers. Young children may not always compensate sufficiently for shorter sleep at night by sleeping for longer periods during the day, further underlying the importance of an early bedtime and obtaining sufficient sleep at night.

5.4.1 Limitations

Several limitations should be considered when interpreting the results. Firstly, parent-reported sleep in early childhood is a limitation. However, this method is common in larger, population-based studies and may even provide a better representation of habitual sleep behaviour than a brief period of objective recording (Sadeh, 2004). This issue is discussed further in the General Discussion. Encouragingly, the mean sleep duration in this sample (11.6 hours per night) is comparable to studies of singletons citing references values for normative sleep behaviour (Blair et al., 2012; Iglowstein et al., 2003). Although bedtime itself is a component from which sleep duration is calculated, the sleep duration variable was dichotomised and neither bedtime nor wake time alone were used to define shorter sleep. Factors associated with shorter sleep also did not show the same pattern of association with bedtime and wake time, indicating that these variables are to some extent under separate influence.

There are no clinical recommendations to define short sleep in early childhood, and as such any cut point will have limitations. The demarcation of short sleep in this study was set at <11 hours per night. Importantly, population means provide some indication of basic sleep requirements in young children. Indeed, part of the rational for using <11 hours is that this value falls below the population mean for nighttime sleep in children aged around 16 months (11.3 hours reported by Blair et al., 2012; see Figure 1.3). However, short sleep in this study was also defined in terms of established health outcomes (obesity being well-documented within the literature). Specifically, the cut point was based on findings from a meta-analysis which examined the impact of sleep on obesity risk in early life. This study found that children under 5 years of age, who slept <11 hours a night, were significantly more likely to be overweight or obese than those children who slept for >11 hours a night (Chen et al., 2008). Consequently, it would be reasonable to argue that the cut point used in this study is sufficient and has clinical relevance. However, it is important to note that the pattern of results were the same when the demarcation of short sleep was set below the 10^{th} percentile of sleep duration (see Appendix G).

The use of twins may be a limitation, and should be considered when interpreting effects related to the number of siblings present in the home. However, it is reassuring that sleep duration is comparable across singletons and twins as this suggests twins are not keeping one another awake at night. There is no strong reason to expect that the predictors of shorter sleep in young children would differ between twins and singletons.

While the present sample was approximately representative of the UK population, the ethnic minority proportion was relatively small. Therefore, it was not possible to examine differences between different ethnic minority groups; this would require a sampling method that focused specifically on ethnic grouping.

5.4.2 Conclusion

Multiple factors within the home environment are associated with shorter sleep in early childhood. Some operate predominantly through later bedtime (ethnicity, maternal education, evening TV), others on earlier wake time (gender), and others have a more general influence on sleep duration (birth weight, older children in the home). In this sample, lower maternal education, ethnic minority status and evening TV viewing, which were among the strongest influences, were associated with shorter sleep through later bedtime. This suggests that bedtime could represent a modifiable target for interventions designed to improve sleep during early life.

Chapter 6 Study 2: Sleep and energy intake in early childhood[10]

6.1 Background and aims

It is important to understand the predictors and pathways to shorter sleep in early life because, as discussed in detail in Chapter 2, shorter nighttime sleep has been strongly implicated in the development of overweight and obesity. Chapter 2 (section 2.2.2) outlined existing experimental studies of sleep restriction in adults, which have consistently shown that excess energy intake can drive weight gain during periods of sleep loss. However, experimental studies involving severe sleep restriction do not necessarily provide information on the longer term impact of sleep on energy intake. Furthermore, findings from adults cannot be assumed to generalise to very young populations as young children have substantially less autonomy over their eating behaviour and food environment than adults (Ventura & Birch, 2008).

While shorter sleep has been more strongly related to weight and weight gain in younger populations (Chapter 2, section 2.1), no studies have investigated the association between sleep and energy intake in very young children (<5 years). This information could help to understand the pathways through which shorter sleep contributes to adiposity in early life.

The aim of the present study was to test the hypothesis that shorter sleep would be related to higher energy intake in early childhood.

[10] A version of this chapter has been published as: Fisher, A., McDonald, L., van Jaarsveld, C. H. M., Llewellyn, C., Fildes, A., Schrempft, S., & Wardle, J. (2014). Sleep and energy intake in early childhood. *International Journal of Obesity, 39,* 1463-1466.

6.2 Methods

6.2.1 Participants

Participants were from the Gemini twin birth cohort, which was described in detail in Chapter 4. The present study analysed data from 1,303 families with complete sleep and dietary data (54% of the baseline sample). Intra-class correlations between twins for sleep and dietary intake were high (sleep: ICC 0.86, 95% CI 0.85 to 0.88; energy intake: ICC 0.87, 95% CI 0.86 to 0.88); therefore to avoid clustering effects by family, one twin was randomly selected from each twin pair. Mothers who provided complete information were slightly older (33.9 versus 32.6 years at twin birth, $P < 0.001$), reported a lower baseline BMI (24.7 vs 25.3, $P < 0.001$), and a larger percentage were university educated (49.1 % versus 39.2%) and from a white British background (88.7% versus 83.9%).

6.2.2 Sleep

Sleep was assessed when the children were approximately 16 months old from parent-reported bedtime and waketime (see Chapter 4, section 4.3.3). Given the lack of consensus on the definition of 'short' versus 'adequate' sleep in the literature (Chen et al., 2008), 5 sleep groups were identified: <10 hours a night, 10 to <11 hours a night, 11 to <12 hours a night, 12 to <13 hours a night and ≥13 hours a night. Categorising sleep at one-hour intervals allowed for comparisons to be made between sleep groups; for example between what might be considered 'optimal' and 'very short' sleeping groups. Categorising sleep in this way was also useful for identifying whether the relationship between sleep and energy intake was linear. Daytime sleep is common in children of this age (Iglowstein et al., 2003) and was assessed with an open-ended question, asking parents to report how long their child usually slept during the day.

6.2.3 Dietary intake

Dietary intake was estimated from diet diaries when the twins were on average 21 months old. The dietary data collection is described further in Chapter 4 (section 4.3.4). In this study, dietary variables examined were; total energy intake (mean daily kcal over 3 days), and mean daily grams of fat, carbohydrate, and protein. The proportion of total energy from fat, carbohydrate and protein was calculated using the Atwater conversion factors (protein: 4 kcal/g, fat: 9 kcal/g and carbohydrate: 3.75 kcal/g) (Food Standards Agency, 2002). In total, 3-day dietary intake data were available for 86.3% of the sample, 9.3% had 2 days, and only 4.4% of children included in the analyses had a single day of dietary data. However, there was no significant difference in energy intake between children with 3 days, versus those with 2 or 1 day of available data. In order to maximise sample size all data were included, however the results did not change when the analyses were repeated including only those children with 3 days of dietary data.

6.2.4 Socio-demographic and weight data

Socio-demographic factors considered as potential confounders were child age, sex, and maternal education. Parents reported their highest education level and this was categorized as: lower (compulsory schooling only; 29% of the sample), middle (some additional school or vocational examinations; 22%), and higher (university education; 49%). Children's age (in months) at sleep questionnaire completion and at diet diary completion were recorded.

Weight was also included as a potential confounder. Gemini families are asked to provide weight measures every 3 months (see Chapter 4, section 4.3.8); weight data recorded between 15 and 24 months of age were selected, and were available for 1094 of the sample (84%).

6.2.5 Statistical analyses

Dietary, eating behaviour, and sleep data were normally distributed. Univariate comparisons for each factor between the 5 sleep groups were made using analysis of variance (ANOVA).

Analysis of covariance (ANCOVA) models were used to compare each dietary variable between the 5 sleep groups adjusting for the factors age, sex, maternal education, and daytime sleep. Polynomial contrasts were used to test for linear associations in the data.

The adjusted models were run with and without weight as a confounder and results were the same, so to maximize sample size ANCOVA models are presented here without adjustment for weight. However, the energy intake model adjusting for weight is presented in Appendix H.

Associations with 24-hour sleep were not examined because daytime sleep has not been associated with obesity risk in prospective studies of children (Bell & Zimmerman, 2010). However, all multivariate models adjusted for daytime sleep.

6.3 Results

Participant characteristics by nighttime sleep duration group are shown in Table 6.1**.** There were no linear associations between any socio-demographic variable and nighttime sleep duration. However, there were slightly more boys in the shorter sleeping groups and slightly more girls in the longer sleeping groups ($P < 0.001$), and maternal education levels varied across sleep groups ($P < 0.001$). There were very slight but significant differences in the age of diet diary completion across sleep groups, with both the shortest and longest sleeping group being slightly younger than the other groups ($P < 0.001$). However, there were no significant differences in daytime sleep duration by nighttime sleep duration. Importantly, weight (kg) and weight SD scores also did not differ significantly by nighttime sleep group (P's > 0.05, Table 6.1).

The unadjusted associations between sleep duration and each dietary variable are shown in Table 6.2. Total energy intake was significantly higher in shorter sleeping groups, with a linear association across sleep durations (P for linear trend = 0.005). Children who slept <10 hours a night consumed on average 1087 kcal/day, while those who slept for ≥13 hours consumed on average 982 kcal/day, a difference of 105 kcal/day. However, sleeping <10 hours or >13 hours a night is relatively uncommon at this age, and the number of children in these groups were small. Therefore, as sensitivity analysis, sleep duration was categorised into three larger groups (<11h, 11-<12h, ≥12), and the linear association between sleep and energy intake was maintained (P for linear trend = 0.025).

Protein intake (grams/day) was not significantly linearly associated with sleep duration (P for linear trend = 0.20). Although differences were small, absolute intakes of fat (g/d) and carbohydrate (g/d) were associated with sleep, with shorter sleepers consuming more (P = 0.02 and P = 0.008 for linear trend, respectively). When expressed as a percentage of total daily calories however, there were no significant differences in macronutrient composition by sleep duration (P's all > 0.05, see Table 6.2).

After adjusting for the factors age, sex, maternal education and daytime sleep, energy intake remained significantly higher in shorter sleeping groups, and the association was linear. In the adjusted model, the difference between the longest and shortest sleeping groups was 102 kcal/day (P for linear trend = 0.008). Figure 6.1 shows the adjusted values for energy intake by nighttime sleep duration.

The associations between total grams of fat and carbohydrate also remained significant after adjustment for age, sex, maternal education and daytime sleep. For fat, the difference between the longest and shortest sleeping groups was 3 grams per day (P for linear trend = 0.02) and for carbohydrate the difference was 10 grams per day (P for linear trend = 0.01). Graphs for the adjusted associations between sleep and fat, carbohydrate and protein intake are shown in Appendix H.

Table 6.1. Participant characteristics by nighttime sleep duration (mean (standard deviation) unless stated otherwise)

	<10 hours	10 - <11 hours	11 - <12 hours	12 - <13 hours	$\geq$13 hours	P (between groups)	P (linear trend)
Number (n=1303)	29	136	602	458	78		
Age at sleep record (months)	15.5 (1.1)	15.8 (0.9)	15.7 (1.1)	15.8 (1.2)	15.7 (1.11)	0.58	0.39
Age at diet diary completion(months)	20.4 (0.7)	20.8 (1.0)	20.7 (1.2)	20.7 (1.4)	20.1 (1.26)	<0.001	0.16
Sex (%) Boy (n=654) / girl (n=649)	52 / 48	58 / 42	54 / 46	42 / 58	43 / 57	<0.001	0.03
Maternal education (%) low (n=380) / mid (284) / high (639)	48 / 21 / 31	38 / 17 / 45	26 / 23 / 51	29 / 20 / 51	35 / 33 / 32	<0.001	0.24
Daytime nap duration (hours)	2.0 (0.9)	1.8 (0.7)	1.8 (0.6)	1.9 (0.6)	1.8 (0.8)	0.80	0.30
Weight (kg)[a]	10.6 (1.4)	11.0 (1.8)	10.9 (1.5)	11.0 (1.6)	10.6 (1.4)	0.16	0.14
Weight SDS[a]	-0.1 (1.0)	0.1 (1.2)	-0.1 (1.1)	0.1 (1.1)	-0.2 (1.1)	0.13	0.12

[a] Weight comparisons are adjusted for exact age when weight data were provided, weight data were available for 84% of the sample

Table 6.2. Dietary data by nighttime sleep duration. Data given as mean (standard deviation)

	<10 hours	10 - <11 hours	11 - <12 hours	12 - <13 hours	≥13h	P (linear trend)
Number (total n=1303)	29	136	602	458	78	
Total energy intake (kcal/d)	1086.8 (208)	1053.4 (43)	1037.4 (182)	1029.1 (187)	981.5 (203)	0.005
Fat (g/d)	43.7 (11.2)	43.6 (10.3)	42.2 (10.1)	42.0 (10.2)	39.2 (10.1)	0.02
Carbohydrate (g/d)	141.5 (31.3)	133.3 (27.2)	132.8 (26.7)	130.8 (25.9)	126.9 (29.5)	0.008
Protein (g/d)	40.8 (10.2)	40.2 (9.2)	39.9 (8.4)	40.3 (8.8)	38.3 (8.2)	0.20
Fat %	36.0 (5.3)	37.2 (5.0)	36.5 (5.0)	36.5 (4.7)	35.9 (4.8)	0.66
Carbohydrate %	49.0 (6.6)	47.6 (5.8)	48.1 (5.6)	47.8 (5.4)	48.5 (5.7)	0.79
Protein %	15.0 (2.6)	15.3 (2.3)	15.4 (2.2)	15.7 (2.2)	15.7 (2.3)	0.06

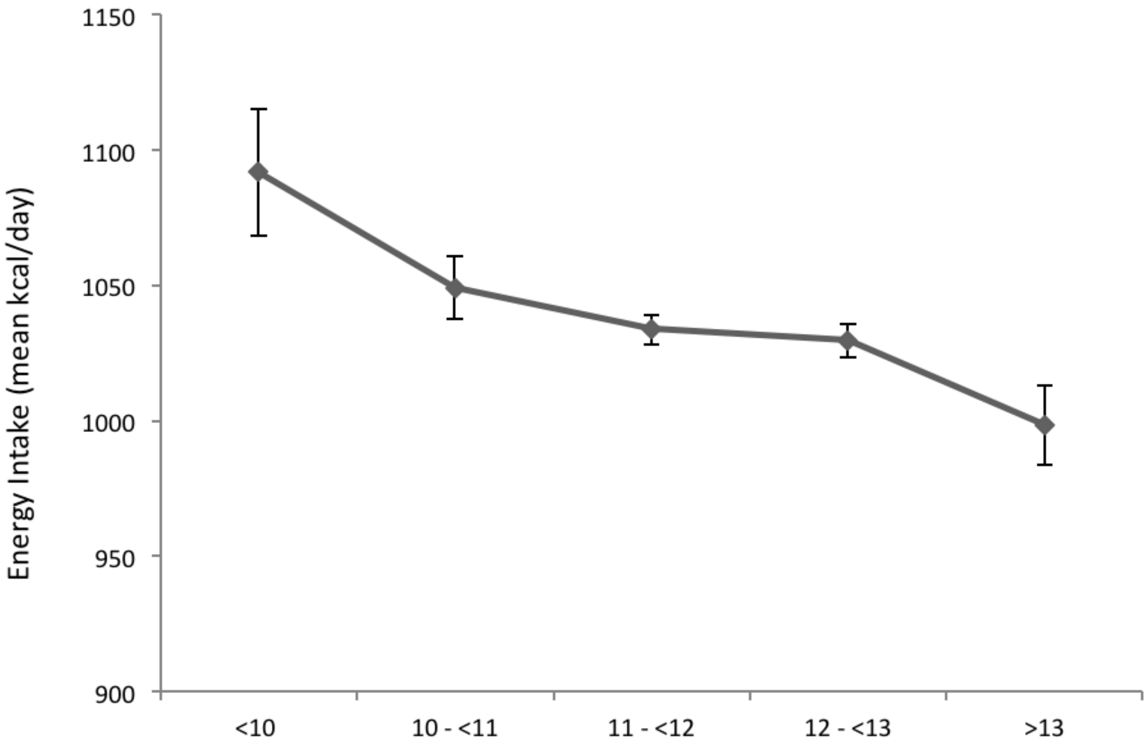

Figure 6.1
Energy intake (kcal/day) by nighttime sleep duration (hours). Values are means adjusted for child age, sex, maternal education and ethnicity. Error bars are standard error.
(P for linear trend =0.003).

6.4 Discussion

This study provides the first evidence in a population sample of young children that shorter sleep is associated with higher energy intake. The observed relationship between sleep and energy intake was linear and emerged before an association with weight was observed, supporting the idea that higher energy intake may be part of the pathway through which shorter sleep influences the development of adiposity in early life.

Experimental studies in adults have consistently shown that sleep restriction promotes excess energy intake and weight gain (see Chapter 2, section 2.2.2 for a review of the literature). However, severe sleep restriction that is enforced under laboratory conditions may not accurately reflect free-living behaviour, particularly that of young children who have much less autonomy over their eating behaviour. Therefore, examining sleep and dietary intake in children, and within a naturalistic setting, has been highlighted as a research priority (Knutson, 2012). Importantly, the present results support findings from experimental studies in demonstrating that shorter nighttime sleep is associated with increasing energy intake. This association was observed in a population sample of young children with diet and sleep reported under free-living conditions, and prior to any relationship between sleep and weight.

There have been few comparable studies in children and none in young children under 5 years of age. One cross-sectional study in 240 adolescents reporting diet from a 24-hour food recall found that those children sleeping less than 8 hours a night (measured from wrist-worn actigraphy) consumed approximately 10% more calories than those sleeping ≥8 hours a night, and consumed a higher proportion of calories from fat (Weiss et al., 2010). The present results add to these findings by demonstrating that associations between sleep and energy intake exist across the spectrum of sleep duration, and among very young children with limited dietary autonomy.

Only one experimental study in school-aged children has examined the impact of sleep loss on energy intake. This study found that when children decreased their sleep duration to 7 hours per night, their daily energy intake increased by 8% (134 kcal per day), and they weighted 0.24 kg more after 1 week (Hart et al., 2013). In the present study, the shortest sleepers (<10 hours) consumed 50 kcal more each day than children obtaining what might be considered an 'optimal' amount of nighttime sleep (11 to <12 hours). While this difference is relatively small, it equates to around 5% of the mean daily energy intake in the sample, and is proportionately comparable to that observed in the aforementioned studies by Weiss et al. (2010) and Hart et al. (2013). Indeed, small-sustained changes over time have the potential to shift the population distribution of overweight and obesity (Boyd & Swinburn, 2011).

In the present study, intake of fats and carbohydrates were significantly higher in shorter sleepers, but this might be expected from the higher energy intake in these groups. Importantly, when considered as a proportion of total energy intake, there were no differences in dietary composition by sleep duration. Rather, children who slept for shorter periods at night tended to eat more overall. However, studies in older age groups have previously identified sleep-related differences in dietary intake. Specifically, shorter sleep has been associated with more unfavorable dietary patterns, including a higher consumption of, or preference for, fat (Grandner et al., 2010; Sato-Mito et al., 2011; Shi, McEvoy, Luu, & Attia, 2008; Weiss et al., 2010), and a lower intake of fruit and vegetables (Garaulet et al., 2011; Moreira et al., 2010; Westerlund et al., 2009). Shorter sleep has also been linked with differences in eating behaviour including increased snacking and eating at more unconventional hours (Kim, DeRoo, & Sandler, 2011; Nedeltcheva et al., 2009; Nishiura, Noguchi, & Hashimoto, 2010; Weiss et al., 2010). In early childhood, parents largely determine the context of feeding, so the impact of sleep on the amount, composition and distribution of energy intake may change between childhood and adolescence with increasing dietary autonomy.

Within this study it was not possible to tell what was driving excess energy intake in shorter sleepers. For example, it is not known whether the increased energy intake was a physiological effect of shorter sleep, or the result of shorter-sleeping children being awake longer and having more time to eat. Some research in adults has suggested that deregulation of the appetite hormones leptin and ghrelin may stimulate energy intake, while other work suggests it may be a result of an increased responsiveness to food stimuli or even a behavioral response to having more time available to eat (this evidence is discussed in detail in Chapter 2, section 2.4). Importantly, very young children do not yet have the physical or volitional autonomy to make decisions about when or how much to eat, so more research is needed to understand the mechanisms by which shorter sleep encourages energy intake in very early life. It would be informative for future work to investigate how and when shorter sleeping children in this study obtained additional calories. This may help to better understand the pathways by which shorter sleep encourages energy intake at this age.

6.4.1 Strengths and limitations

A key strength of this study is the use of detailed dietary data in a population-based cohort; the dietary data used in this study is the largest of data-set of its kind that currently exists for toddlers in the UK. The hypothesis presented in this study is that shorter sleep impacts energy intake. Despite the strength of the dietary data, the cross-sectional nature of this study means it is not possible make conclusions about causality, or exclude the possibility that increasing energy intake impacted subsequent sleep duration. However, examining associations between sleep and dietary intake at a very young age, and before relationships between sleep and weight emerge does strengthen the argument for a causal role. That sleep was not associated with weight at this young age also reduces the possibility that already bigger children were simply eating more.

The reliance on parent-reported sleep duration is a limitation, but this was the only feasible method to assess child sleep in Gemini, due to funding restrictions. The limitations associated with parent-reported sleep are discussed in detail in the General Discussion. However, it is important to consider that by reducing measurement error, objective measures of sleep would most likely increase the strength of the sleep and energy intake association reported here.

In order to examine linear trends within the dietary data, and to reflect the various cut-points used to define ‘short’ sleep in the paediatric sleep and weight literature, sleep duration was categorised into 5 groups. Although sleeping <10 hours or >13 hours a night is not typical at this age, and the number of participants in these groups were small, a sensitivity analysis demonstrated that the significant linear trend in energy intake was maintained when sleep was categorised into three larger groups (<11 hours, 11 to <12 hours, ≥12 hours; P = 0.025), suggesting that this was a robust effect.

The Gemini sample is a twin cohort, albeit this study used data from only one child per family. There are important differences between twins and singletons; in particular, twins tend to be born smaller and remain smaller in early life relative to singletons (Buckler & Green, 2004). The limitations associated with using a twin sample are considered in more detail in the General Discussion. However, it is encouraging that the average daily caloric intake in this sample is comparable to reference values for singleton children of this age (see Chapter 4, Table 4.5) (Lennox et al., 2011). Furthermore, the mean nighttime sleep duration reported here (11.6 hours per night) is comparable to studies of singletons reporting references values for nighttime sleep (11.3 hours, Figure 1.3) (Blair et al., 2012; Iglowstein et al., 2003). There is also no reason to expect that the relationship between sleep and energy intake would differ between twins and singletons, although this possibility cannot be ruled out.

There were small but significant socioeconomic and ethnic differences between families who provided complete data and those who did not. However, the magnitude of the observed differences was small, and again, there is no strong reason to suspect that associations between sleep and energy intake would differ by socioeconomic group. Nevertheless, these associations should be examined in larger samples of ethnically diverse populations.

6.4.2 Conclusion

Shorter nighttime sleep duration is associated with higher energy intake in early childhood, before differences in weight have emerged. A higher energy intake is a plausible mechanism through which shorter sleep may contribute to adiposity in early life.

Chapter 7 Study 3: Sleep and nighttime energy consumption[11]

7.1 Background and aims

The findings from Study 2 (Chapter 6) strongly implicate energy intake as a pathway through which shorter sleep contributes to adiposity in early life. However, it is not known how and when additional calories were consumed. This information could help to understand the pathways by which shorter sleep leads to excess energy intake in young children.

Experimental studies in adults have shown that excess energy intake during sleep loss is consumed predominantly at night during the hours of extended wakefulness, and often from energy-dense snacks (this evidence is reviewed in detail in Chapter 2, section 2.2.2). This is important as the timing of food intake has itself been implicated in weight regulation, with later eating times associated with higher BMI, poorer weight loss outcomes, and a higher risk of metabolic abnormalities (Corbalán-Tutau et al., 2012; Garaulet et al., 2013; Romon, Edme, Boulenguez, Lescroart, & Frimat, 1993).

Young children have limited autonomy over their feeding behaviour and food environment (Ventura & Birch, 2008), and therefore cannot be assumed to show the same temporal patterning of intake in response to shorter sleep as adults. Study 1 in Chapter 5 demonstrated that shorter-sleeping children tend to have a later bedtime rather than an earlier wake time (McDonald, Wardle, Llewellyn, van Jaarsveld, & Fisher, 2014), which could suggest that shorter sleeping young children may also be susceptible to overconsumption in the evening/nighttime period. However, no studies have examined whether habitually shorter sleeping young children consume more at night.

The aim of the present study was to test the hypothesis that energy intake would be specifically higher in the evening in shorter-sleeping children, by examining the temporal

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patterning of energy intake in relation to sleep duration. A secondary aim was to determine whether the additional calories were from meals, snacks or drinks.

7.2 Methods

7.2.1 Participants

Participants were 1,278 families participating in the Gemini study, described in detail in Chapter 4. Data were from families who had provided complete information on diet and sleep (53.2% of the baseline sample). Children with complete data had mothers who were slightly older (34.6 versus 32.5 years at twin birth, $P < 0.001$), had a slightly lower BMI at baseline (24.7 versus 25.6, $P < 0.001$), were more highly educated, and who were more likely to be from a white ethnic background (P's all < 0.001). As previously stated, intra-class correlations between twins for the sleep and dietary data were high (sleep: ICC 0.86, 95% CI: 0.85 to 0.88; energy intake: ICC 0.87, 95% CI: 0.86 to 0.88); therefore to avoid clustering effects by family, one twin was randomly selected from each twin pair.

7.2.2 Sleep

As described in Chapter 4 (section 4.3.3) sleep was assessed when the children were approximately 16 months old (mean 15.7, SD 1.1 months) from parent reported bedtime and wake time. Given the uncertainty in the literature over how best to define 'short' sleep (Matricciani, Olds, Blunden, Rigney, & Williams, 2012), 5 sleep groups were identified: <10 hours a night, 10 to <11 hours, 11 to <12 hours, 12 to <13 hours, and ≥13 hours a night, which allowed a test of linear associations. The same groupings were used in Study 2 to examine linear associations between sleep and daily energy intake at the same age. Associations with 24-hour sleep were not examined because daytime has not been associated with obesity risk in prospective studies of children (Bell & Zimmerman, 2010), although daytime sleep was adjusted for in all multivariate models.

7.2.3 Dietary intake

Dietary data, described in Chapter 4 (section 4.3.4), were collected when children were on average 21 months old (mean 20.7, SD 1.3 months). In total, 86.2% of children included in the analyses had complete data for 3 days of intake, 9.3% had 2 days, and only 4.5% of children had 1 recoded day of intake. However, no significant difference in energy intake was observed between children with 3 days, versus those with 2 or 1 day of available data. In order to maximise sample size, all children with at least 1 day of dietary data were included in the analyses.

In this study, dietary variables examined were total energy intake (mean kcal/day) and energy intake from meal, snack and drink occasions (mean kcal/day from each type of occasion). The classification of energy intake into meal, snack and drink occasions is described in detail in Chapter 4 (section 4.3.4). Briefly, all food and drink items within a single time entry were defined as one eating occasion. Each occasion was then coded as a meal or snack using a food-based classification system (Macdiarmid et al., 2009). An eating occasion including a meal food item (e.g. meat, fish, eggs, cooked vegetables) was coded as a meal, while an occasion with one or more snack items (e.g. a cookie, crisps, yogurt, raw vegetables) without a meal item was coded as a snack. A drink occasion was defined as a drink consumed without any food items. The coding frame is presented Appendix C.

7.2.4 Socio-demographic and weight data

Sex, birth weight, gestational age, and maternal education were included as potential confounders. This data was collected in baseline questionnaires (see Chapter 4, section 4.3). For the purposes of this study, maternal educational was dichotomised into lower (no university level education; 51%) and higher (university education; 49%). As described in Chapter 4, Gemini families are asked to provide weight measures every 3 months. Weight data recorded between 15 and 24 months of age were identified, and were available for 84% of the sample.

7.2.5 Statistical analyses

7.2.5.1 The temporal patterning of energy intake

Dietary and sleep data were normally distributed. To examine the temporal patterning of energy intake, the 24-hour dietary day was divided into four periods designed to cover the range of conventional meal/snack intakes for young children in the UK: morning (6:00 am to < 10:00 am), daytime (10:00 am to < 3:00 pm), afternoon/evening (3:00 pm to < 19:00 pm) and night (7:00 pm to < 6:00 am). The start of the nighttime period was fixed according to the average bedtime in the sample (7:12 pm), and the end of this period was slightly before the average wake time of 6:50 am.

To test whether sleep was associated with temporal variations in energy intake, univariate analysis of variance (ANOVA) compared energy intake between the 5 sleep groups for each time period. Tests of a linear association (using polynomial contrasts) were used to determine whether energy intake increased or decreased with sleep duration for a given time period.

7.2.5.2 Partitioning energy intake into meals, snacks and drinks

To identify how additional calories were being consumed, univariate ANOVA's using tests of a linear association were run to test whether sleep was associated with differences in energy intake from meals, snacks or drinks for any time period where shorter sleepers reported higher energy intake.

All univariate analyses were repeated adjusting for age, sex, birth weight, gestational age, maternal education, and daytime sleep duration in analyses of covariance (ANCOVA) with polynomial contrasts (testing for a linear association). Daytime sleep is common in children of this age (Blair et al., 2012), and was included as a potential confounder in case shorter nighttime sleepers were compensating with more sleep during the day.

7.3 Results

Participant characteristics by nighttime sleep duration are presented in Table 7.1. There were no linear associations between any socio-demographic variable or daytime sleep and nighttime sleep duration. However, the shorter sleeping groups had slightly more boys, and a smaller proportion of their mothers were university educated. Shorter sleepers had both a later bedtime and an earlier wake time. Nighttime sleep was not associated with weight at this age (these findings are presented and discussed in Chapter 6, Study 2).

7.3.1 The temporal patterning of energy intake

Dietary data by sleep duration are presented in Table 7.1**.** Analysis of the temporal patterning of energy intake showed that energy intake in the morning (6:00 am to < 10:00 am) and daytime (10:00 am to < 3:00 pm) did not differ significantly by sleep duration.

Energy intake in the afternoon/evening (3:00 pm to < 7:00 pm) was significantly *lower* in the shorter sleeping groups in a linear fashion ($P = 0.023$), although the absolute differences were very small. Children sleeping <10 hours a night consumed 319 kcal in the evening, while children in the longest sleeping group consumed on average 360kcal; a difference of around 41 kcal.

At night (7:00 pm to < 6:00 am), energy intake was significantly higher in the shorter sleepers in a linear fashion ($P < 0.001$). Children in the shortest sleeping group (<10 hours a night) consumed on average 166 kcal at night, while those in the longest (≥13 hours) consumed 46 kcal; an average difference of 120 kcal. Over 90% of calories consumed during the nighttime period, across all sleep groups, were consumed before 12 midnight (range for energy consumed 12:00 pm to 6:00 am was 3-10 kcal across the sleep groups).

7.3.2 Partitioning energy intake into meals, snacks, drinks

Across all drink occasions recorded, 76.8% were milk drinks, and 98.6% of all calories in drink occasions came from milk drinks. Partitioning energy intake at night into energy obtained from meals, snacks and drinks showed that shorter sleep was associated with higher intake from all types of occasions (P's <0.001). However, the calorie intake from meal and snack occasions was small, amounting only to a 20 kcal difference from shortest to longest sleepers (see Figure 7.1). The largest difference in nighttime calories was from drinks, which in this sample were almost all milk drinks (98.6%). On average, children in the shortest sleeping group consumed 118 kcal from drinks at night, while those in the longest consumed 40 kcal, with a linear pattern in between. Adjusted values for calories consumed at night by eating occasion are presented in Figure 7.1.

Associations between sleep and energy intake (both by time of day and eating occasion) were unchanged after adjusting for age, sex, birth weight, gestational age, maternal education and daytime sleep. Adjusted values are presented in Appendix I.

Table 7.1. Unadjusted dietary data by nighttime sleep duration (mean (standard deviation))

	< 10 hours	**10 - < 11 hours**	**11 - < 12 hours**	**12 - < 13 hours**	**≥ 13 hours**	**p (linear trend)**
Total energy intake (mean kcal per day)	1088.17 (195.35)	1046.83 (198.44)	1035.39 (188.24)	1025.83 (189.39)	1006.05 (202.39)	0.020
Total energy intake by eating occasion (mean kcal per day)						
Meals	691.65 (179.39)	674.20 (194.46)	715.99 (186.83)	707.48 (188.45)	703.12 (199.09)	.473
Snacks	176.15 (137.50)	165.17 (115.51)	150.73 (101.38)	149.49 (110.97)	140.84 (98.20)	.053
Drinks only	220.37 (154.09)	207.46 (120.26)	168.67 (111.02)	168.48 (119.39)	162.09 (131.42)	.002
Total energy intake by time of day (mean kcal per day)						
Morning	253.43 (81.50)	275.32 (97.10)	285.65 (93.26)	277.15 (94.67)	260.06 (86.17)	0.698
Day	349.77 (116.85)	329.67 (94.14)	326.34 (96.71)	334.56 (95.52)	340.11 (120.76)	0.724
Afternoon/Evening	319.40 (129.86)	328.32 (116.99)	349.66 (114.06)	356.93 (114.78)	359.94 (119.80)	0.023
Night	165.55 (88.84)	113.52 (92.86)	73.74 (79.64)	57.19 (75.72)	45.94 (71.98)	<0.001
Total energy intake at night by eating occasion (mean kcal per night)						
Meals	25.44 (50.44)	9.59 (32.22)	6.77 (33.25)	4.68 (33.45)	5.04 (25.69)	0.001
Snacks	22.10 (37.13)	11.97 (43.64)	3.61 (17.28)	3.24 (22.39)	0.89 (7.41)	<0.001
Drinks only	118.02 (79.94)	91.96 (75.91)	63.37 (71.01)	49.28 (62.86)	40.00 (63.66)	<0.001

Table 7.1. Participant characteristics by nighttime sleep duration (mean (standard deviation) unless otherwise stated)

	< 10 hours	10 - < 11 hours	11 - < 12 hours	12 - < 13 hours	≥ 13 hours	p (linear trend)	p (between groups)
Total (n = 1278)	36	129	599	437	77		
Age at sleep record (months)	15.67 (1.10)	15.78 (1.08)	15.73 (1.07)	15.75 (1.17)	15.67 (0.96)	0.919	0.944
Age at diet diary record (months)	20.50 (0.94)	20.73 (0.98)	20.74 (1.14)	20.74 (1.25)	20.39 (0.98)	0.653	0.101
Sex (%)							
Boy (n=619)	50.0	59.7 [b]	52.8 [b]	39.4	46.8		
Girl (n=659)	50.0	40.3 [b]	47.2 [b]	60.6	53.2	0.194	<0.001
Birth weight (kg)	2.31 (0.51)	2.38 (0.55)	2.49 (0.53)	2.47 (0.52)	2.38 (0.53)	0.314	0.050
Gestational age (weeks)	35.92 (2.80)	36.05 (2.97)	36.29 (2.46)	36.30 (2.18)	35.87 (2.30)	0.874	0.445
Maternal education (%)							
Low (n=499)	66.7	43.4	35.4 [c]	38.2 [c]	51.9		
High (n=779)	33.3	56.6	64.6 [c]	61.8 [c]	48.1	0.086	<0.001
Daytime sleep (hours)	1.93 (0.86)	1.92 (0.76)	1.83 (0.61)	1.89 (0.69)	1.79 (0.80)	0.258	0.441
Bedtime	9:08 (1:14)	7:46 (0:51)	7:33 (0:30)	6:58 (0:31)	6:33 (0:34)	<0.001	<0.001
Wake time	6:15 (1:08)	6:12 (0:47)	6:38 (0:30)	7:11 (0:32)	7:49 (0:42)	<0.001	<0.001
Weight (kg)[a]	10.22 (1.20)	10.87 (1.67)	10.93 (1.58)	10.90 (1.50)	10.87 (1.50)	0.063	0.213

[a] weight data available for 84% of the sample; [b] significantly different from 12-13 hours group; [c] significantly different from < 10 hours group

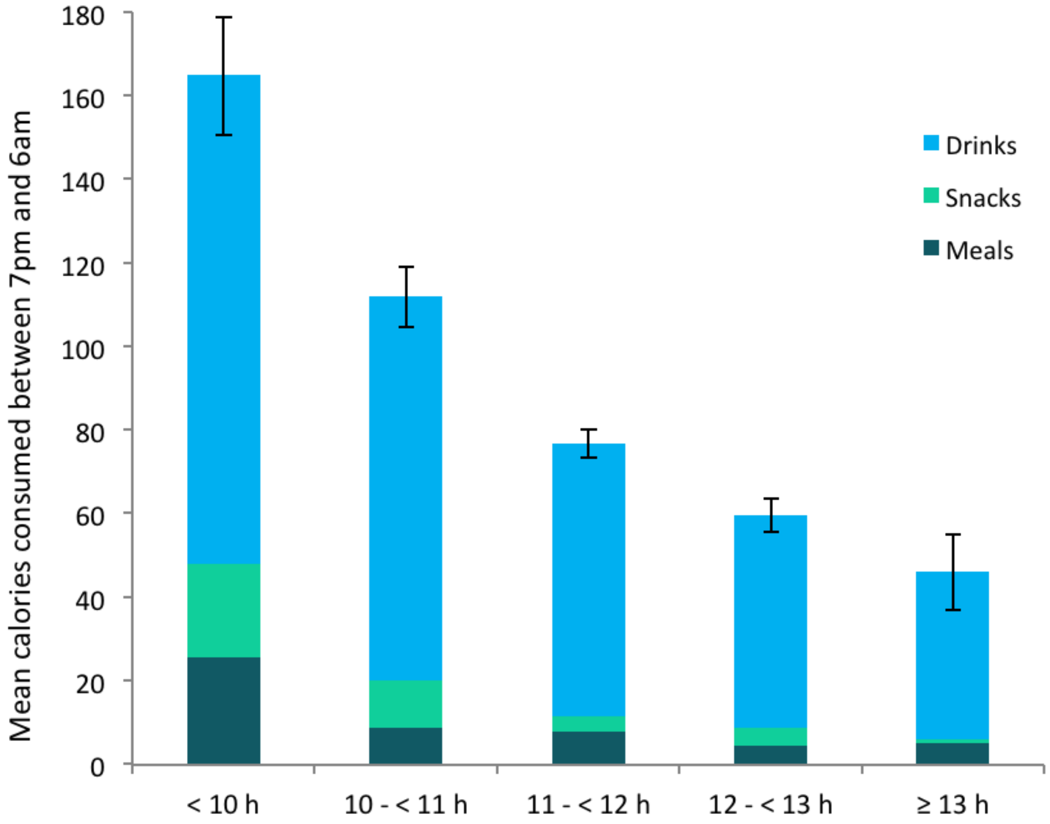

Figure 7.1

Energy intake at night (mean (SE)) obtained separately from meals, snacks and drinks by nighttime sleep duration (hours). Values are adjusted for age, sex, birth weight, gestational age, maternal education and daytime sleep (P for linear trend < 0.001).

7.4 Discussion

This study shows that shorter sleep in young children is associated with higher energy intake between the hours of 7:00 pm and 6:00 am, and that the majority of additional intake comes from milk drinks consumed before midnight. This finding persisted after adjustment for multiple potential confounders. Nighttime feeding of milk drinks may be an important means through which shorter sleep contributes to excess energy intake in early life, and provides a potential target for intervention.

This is the first study to demonstrate that sleep is associated with temporal variations in energy intake in children under the age of 5 years. The findings are consistent with experimental data in adults and older children which have shown that consecutive nights of sleep restriction encourage the consumption of additional energy at night (Hart et al., 2013; Markwald et al., 2013; Spaeth et al., 2013). However, the results are important in showing that temporal variation in energy intake exists in habitually short sleepers, and not only after experimentally induced sleep restriction. Identifying temporal patterning in energy intake among young children with limited feeding autonomy is also important given that associations between sleep and weight appear stronger in paediatric populations (Cappuccio et al., 2008). Supporting the causal association, this study demonstrated a linear association between sleep duration and nighttime energy intake. Average nighttime intake went from 166 kcal in the shortest sleepers (<10 hours), through 114, 74, and 57 to 46 in the longest sleepers (>13 hours). Although the nighttime period extended to 6 am, almost all nighttime calories were consumed before midnight (over 90% across the 5 sleep groups). Shorter sleepers also consumed slightly less in the afternoon/evening period, but these differences were comparatively small, and did not compensate for the higher energy intake at night.

The finding of higher nighttime energy intake among shorter sleepers was seen at an age when the association between shorter sleep and weight had not yet been observed (these results were presented in Chapter 6, Study 2). This has the advantage of precluding the possibility of confounding by weight status, although it also means that longer-term follow up is needed to demonstrate that the nighttime energy intake observed in this study is a mechanism through which shorter sleep contributes to weight gain in early life. Activity levels or energy expenditure were not measured in this study, but an important observation from experimental studies is that excess intake during sleep restriction is not adequately offset by energy expenditure (Hart et al., 2013; Markwald et al., 2013; Spaeth et al., 2013). As energy intake is lowest during sleep, being awake rather than asleep can be assumed to incur some energetic cost (Markwald et al., 2013), although total energy expenditure among shorter sleepers may be reduced by fatigue (Schmid et al., 2009).

The results show that shorter sleeping children had both a later bedtime and an earlier wake time, but only consumed more than longer sleepers in the nighttime period. It may be that having more time to eat, particularly at night, facilitates excess energy intake. This may help to explain why both short sleep and later sleep timing have been associated with weight gain in prospective studies of children (Scharf & DeBoer, 2014). Indeed for children sleeping <10 hours a night, nighttime consumption alone accounted for 15.2% of total daily energy intake. This is strikingly comparable to the findings on nighttime intake in adults exposed to enforced sleep loss, which has ranged from 14% to 22% of daily energy intake (Spaeth et al., 2013).

Identifying that shorter sleeping children consume more at night is important because the timing of food intake has been implicated in weight regulation, with later eating times being associated with adiposity, poorer weight loss outcomes, and a higher risk of metabolic dysfunction (Baron, Reid, Kern, & Zee, 2011b; Garaulet et al., 2013; Jakubowicz, Barnea, Wainstein, & Froy, 2013). This may occur because diet-induced thermogenesis is lower in the evening and night than in the morning (Romon et al., 1993). The fact that the additional energy intake in shorter sleepers is consumed predominantly at night may therefore contribute both to the development of adiposity and to its consequences.

In this sample, the majority of nighttime calories were obtained from drinks, of which over 98% were from milk drinks. While this study did not assess why parents were giving their children additional drinks at night, one possibility is that hunger levels may be endogenously higher in the evening (Scheer et al., 2013), so parents could have been responding to perceived hunger. Alternatively, because provision of milk drinks is a common parental strategy to help young children initiate sleep (Sadeh, Mindell, Luedtke, & Wiegand, 2009), parents could have been providing milk to soothe their child before sleep or during the night, but another possibility is that mothers are 'filling up' their children in order to get them to sleep through the night. Although commonly used by parents to promote good sleep, feeding at night is usually associated with poorer sleep outcomes in children under the age of 5 years (Touchette et al., 2005b), possibly because this practice prevents the child from developing the capacity to self-soothe (Sadeh, Tikotzky, & Scher, 2010). Habits form when behaviours are repeated frequently in a stable context, with the context alone becoming sufficient to cue to the behaviour (Lally & Gardner, 2013). Routinely providing milk before bed or as part of a bedtime routine may therefore result in nighttime calorie consumption becoming habitual, and dissociated from the child's physiological requirements. While no studies have examined whether nighttime consumption in routinely short sleepers is driven by habit, food intake in night shift workers has been shown to be more strongly influenced by habit and food availability at night than by appetite (Waterhouse, Buckley, Edwards, & Reilly, 2003).

Feeding before bed or during the night may be good practice in infants who are exclusively milk-fed, but after weaning this should no longer be necessary (Touchette et al., 2005b). This study demonstrates that providing milk drinks at night results in shorter sleepers consuming more energy; potentially contributing to later weight gain. If this practice also operates to dissociate feeding from hunger, and doesn't aid sleep, parents could be encouraged to use alternative methods to help initiate and consolidate nighttime sleep in young children.

7.4.1 Strengths and limitations

This study benefited from detailed food diaries that recorded multiple days of dietary intake and included a timed entry for each eating occasion. The use of parent-reported of sleep behaviour is a limitation, but this method is commonly used in large-scale cohorts where objective measurements are not feasible (Chen et al., 2008). Encouragingly, the average nighttime sleep duration in this sample (11.6 hours) is comparable to published reference values for this age group (Iglowstein et al., 2003).The limitations associated with parent-reported nighttime sleep are discussed further in the General Discussion.

As in Study 2 (Chapter 6), sleep duration was categorized into 5 groups at 1-hour intervals specifically to examine linear trends within the dietary data. However, sleeping <10 hours or ≥13 hours a night is relatively uncommon in children of this age, and the numbers of children represented in these groups were small. Nonetheless, it is important to note that when sleep duration was treated as a continuous variable, all associations with nighttime intake remained significant.

It may be a limitation that there was a slight time lag between the sleep (16 months) and dietary assessments (21 months). However, nighttime sleep duration is relatively stable at this age (see Figure 1.3; Blair et al., 2012), and patterns of sleep behaviour tend to correlate over the first 3 years of life (Byars, Yolton, Rausch, Lanphear, & Beebe, 2012), so it is likely that sleep duration would be similar at the two times. Although this period of time is too short to be considered truly prospective, it does support the direction of the hypothesised relationship.

A complex relationship exists between sleep, circadian rhythms and energy regulation (Gonnissen, Hulshof, & Westerterp-Plantenga, 2013), and it is not possible to exclude the possibility that the increased energy intake at night disturbed subsequent sleep initiation or consolidation (Al-disi, Al-daghri, Khanam, Al-othman, & Al-saif, 2010), particularly given that sleep disturbances and feeding problems can coexist in early life (Tauman et al., 2011). However, it is encouraging that the findings were consistent with results from experimental studies of sleep restriction (Hart et al., 2013; Markwald et al., 2013).

There were some differences between families that provided complete data and those that did not. Specifically, children with complete data had mothers who were more highly educated, had a lower BMI, and were more likely to be from a white ethnic background. This could limit the generalisability of the findings, so replication in a more diverse sample is required. Although participants were from a cohort of twins, this study included data from one twin selected at random from each pair. There is no reason to expect that any of the observed associations between sleep and eating behaviour would differ in singletons. It is also reassuring that the average daily caloric intake in this sample is comparable to reference values for children of this age (Lennox et al., 2011).

7.4.2 Conclusion

This study shows that shorter sleeping children consume more energy than longer sleepers predominantly after 7:00 pm, and the majority of the extra calories come from milk drinks consumed before midnight. This could lead to simple guidance to parents to avoid excess energy intake in nighttime drinks for young children.

Chapter 8 Study 4: Nighttime sleep and hedonic eating[12]

8.1 Background and aims

Evidence presented in this thesis and discussed in detail in the literature review in Chapter 2 points to food intake as the primary pathway through which insufficient sleep may increase the risk of adiposity in early life. Studies 2 and 3 identified an inverse relationship between sleep duration and energy intake. These findings are supported by experimental and epidemiological data in older children and adults which have also shown that shorter nighttime sleep is associated with increased energy intake and weight gain (Golley, Maher, Matricciani, & Olds, 2013; Markwald et al., 2013; Spaeth, Dinges, & Goel, 2013).

A necessary progression is to understand the mechanisms through which shorter sleep may affect food intake. Recent observations suggest that sleep may impact energy intake via hedonic rather than homeostatic pathways (see Chapter 2, section 2.4 for a full discussion and review of the literature). For example, neuroimaging data show that sleep deprivation increases activity in brain reward centres in response to images of palatable food, as well as 'desire to eat' (Greer, Goldstein, & Walker, 2013). Furthermore, experimental studies in adults have shown that controlled sleep curtailment in the context of *ad libitum* energy intake increases preference and consumption of energy-dense foods, without any evidence of change in metabolic (homeostatic) signals (Markwald et al., 2013).

To date, most studies have investigated the acute effects of sleep deprivation. There have been few studies examining relationships between free-living sleep and hedonic eating. Data are particularly limited in paediatric populations, despite this being the stage at which associations between sleep and weight appear most consistent (Cappuccio et al., 2008; see Chapter 2, section 2.1). One small study in 56 children aged 5 to 12 years showed that shorter sleep was associated with lower scores on a scale of 'external eating', a construct

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with considerable overlap with hedonic processes, but associations between sleep and weight were not reported (Burt et al., 2013).

In the present study, the primary hypothesis was that shorter sleep at age 5 years would be associated with hedonic eating (indexed with food responsiveness). In a subset of participants with weight data at 5 years, this study tested the hypothesis that hedonic eating would partly mediate the association between sleep duration and weight. As a secondary analysis, this study also examined whether sleep duration was associated with homeostatic eating (indexed with satiety responsiveness) to test whether any sleep-eating behaviour association was general rather than specific to hedonic eating.

8.2 Methods

8.2.1 Participants

Participants were from 1,008 families (42% of the baseline sample) from the Gemini twin birth cohort, which is described in detail in Chapter 4. The present study used data collected in 2012, when the children were on average 5 years old. Data were from families who had provided complete information on their children's sleep and appetite at this age. Mothers who provided complete data were slightly older (33.9 versus 32.3 years), reported a slightly lower BMI at baseline (24.7 versus 25.4), were more highly educated, and more likely to be from a white ethnic background (P's all <0.001). To avoid clustering effects, one child from each twin pair was randomly selected for the analysis.

At this age, complete height and weight data were available for 494 children (21% of baseline sample). Mothers who provided this information were more likely to be university educated, but further to this there were no differences between the study sample and the sample with BMI data on any key variables including birth weight, sleep duration, satiety responsiveness or food responsiveness. Comparison data for these variables are given in Appendix J.

8.2.2 Measures

8.2.2.1 Sleep

Nighttime sleep duration was calculated from parent-reported bedtime and wake time at age 5. Sleep was then categorised as shorter (<11 hours), adequate (11-12 hours) or longer (>12 hours). These categorisations were based on population means for sleep at this age (Blair et al., 2012). Given the smaller sample size at this age it was not possible to divide the sample into smaller groups at 1-hour intervals as in studies 2 and 3. However, categorising sleep in this way allowed for comparisons to be made previous studies presented in this thesis, the sleep-weight literature, and also allowed for tests of a linear association.

8.2.2.2 Eating Behaviour

Hedonic eating was assessed with the food responsiveness scale of the Child Eating Behaviour Questionnaire (CEBQ) (Wardle et al., 2001). Satiety responsiveness was also included and assessed with the satiety responsiveness scale of the CEBQ as an indicator of 'homeostatic eating'. The CEBQ was described in detail in Chapter 4 (section 4.3.7). Briefly, it is a validated 35-item instrument designed to assess a range of appetitive traits implicated in the regulation of body weight (Wardle et al., 2001). The food responsiveness scale has 5 items assessing the degree to which a child expresses a desire for food, particularly in response to palatable food cues (e.g. my child is always asking for food). The satiety responsiveness scale has 5 items assessing the degree to which a child tends to stop eating (or doesn't initiate eating) according to their perceived fullness (e.g. my child cannot eat a meal if s/he has had a snack just before). Both the food responsiveness and satiety responsiveness scales have good internal consistency and reliability, and have been validated against objective measures of food intake in childhood (Carnell & Wardle, 2007; Wardle et al., 2001).

8.2.2.3 Body Mass Index standard deviation score

As described in Chapter 4 (section 4.3.8) Gemini families were provided with electronic weighing scales and height charts when the children were 24 months old, and are asked to provide 3-monthly height and weight measures. Weight and height data included in the analyses were those collected closest to questionnaire completion. If exact 5-year data were not available, data points within 3 months of this age were used. Weight and height data were available for 494 children at age 5. Age- and sex-adjusted body mass index standard deviation scores (BMI-SDS) were calculated using UK 1990 reference data (Cole, Freeman, & Preece, 1995).

8.2.2.4 Socio-demographic characteristics

As outlined in Chapter 4 (section 4.3.10), maternal education was assessed in the baseline questionnaire. Mothers reported their highest level of education, which was categorised into lower (compulsory basic schooling) middle (some additional schooling or vocational qualifications) and higher (university educated). Birth weight was reported by asking parents to photocopy or transcribe health records.

8.2.3 Statistical analysis

Sleep and appetite data were normally distributed. Univariate ANOVA models using tests of a linear association were used to compare food responsiveness and satiety responsiveness between the 3 sleep groups. ANCOVA models were then run to test whether any observed effects were independent of age, maternal education, sex, and birth weight. Models were also adjusted for BMI-SD scores, however doing so substantially reduced the sample size, and therefore adjusted models are presented with and without the inclusion of BMI-SDS.

In the subset of the sample with available weight data, comparison of BMI-SDS between sleep groups was carried out using ANOVA with polynomial contrasts testing for a linear association. ANOVA models were first run to examine the linear relationship between sleep duration and BMI-SDS. To test whether the sleep-weight relationship was attenuated by differences in eating behaviour, any trait significantly associated with sleep in previous models was entered into the ANCOVA model predicting BMI-SDS, adjusting for age, maternal education, sex, and birth weight. Hayes' bootstrapping PROCESS macro for SPSS was used to evaluate mediation (Hayes & Preacher, 2013).

8.3 Results

Participant characteristics are shown in Table 8.1. In total, 1,008 children had complete sleep and appetite data at age 5 (mean 5.2, SD 0.1 years). Average nighttime sleep duration was 11.48 hours (SD 0.66 hours), the average food responsiveness score was 2.84 (0.61), and the average satiety responsiveness score was 2.38 (0.76). BMI-SDS information was available for 494 children, with a mean value of -0.20 (0.96).

Univariate and multivariate associations between sleep and food responsiveness are shown in Table 8.2. There was a significant linear relationship between nighttime sleep duration and food responsiveness, such that shorter sleep was associated with higher food responsiveness at age 5 (P for linear trend = 0.032). These associations were retained after adjusting for age, sex, maternal education, birth weight, and BMI-SDS; the latter adjustment reducing sample size. There was no significant association between nighttime sleep duration and satiety responsiveness (P for linear trend = 0.749).

The results of ANOVA and ANCOVA models predicting BMI-SDS in the subset of the sample with available weight data are given in Table 8.3. As expected, shorter nighttime sleep was associated with higher BMI-SDS (P for linear trend = 0.026). The linear relationship strengthened after adjusting for age, sex, birth weight and maternal education (P for linear trend = 0.015). To test mediation by eating behaviour, food responsiveness was included into the ANCOVA model predicting BMI-SDS. This model attenuated the linear association between sleep and BMI-SDS to borderline significance (P for linear trend = 0.049). The Hayes' PROCESS add-in for SPSS demonstrated the mediation effect via food responsiveness was significant [-0.04 (0.02); 95 %CI, -0.09 to -0.01].

Table 8.1. Participant characteristics given as mean (standard deviation) unless otherwise stated

n	1008
Birth weight	2.46 (0.54)
Maternal education (%) low/ mid/ high	27.4 / 22.1 / 50.5
Sex (%) male / female	49.4 / 50.6
Nighttime Sleep (hours)	11.48 (0.66)
Appetite	
Satiety Responsiveness	2.38 (0.76)
Food Responsiveness	2.84 (0.61)
BMI-SDS[a]	-0.21 (0.96)

[a]Data available for n = 494 children

Table 8.2. ANOVA and ANCOVA models for the association between appetitive traits and nighttime sleep duration. Data given as mean (standard error).

Nighttime sleep duration	<11h	11-12h	>12h	P (linear trend)
Univariate models				
Food responsiveness	2.53 (0.08)	2.36 (0.03)	2.35 (0.04)	0.032*
Satiety responsiveness	2.80 (0.07)	2.89 (0.02)	2.80 (0.03)	0.749
Multivariate models[a]				
Food responsiveness	2.55 (0.07)	2.36 (0.03)	2.35 (0.05)	0.022*
Satiety responsiveness	2.82 (0.06)	2.88 (0.03)	2.76 (0.04)	0.372
Multivariate models[b]				
Food responsiveness	2.51 (0.09)	2.32 (0.04)	2.28 (0.60)	0.038*
Satiety responsiveness	2.85 (0.08)	2.89 (0.03)	2.75 (0.05)	0.229

[a] Adjusted for age, sex, birth weight, maternal education
[b] Adjusted for all covariates and BMI-SDS
*P < 0.05

Table 8.3. ANOVA and ANCOVA models for association between sleep and BMI-SDS, adjusting for food responsiveness and satiety responsiveness. Data given as mean (standard error).

Nighttime sleep duration	<11 hours	11-12 hours	>12 hours	P (linear trend)
BMI-SDS				
Univariate	0.01 (0.15)	-0.15 (0.05)	-0.35 (0.10)	0.026*
Multivariate models				
Model 1	0.05 (0.14)	-0.16 (0.06)	-0.36 (0.09)	0.015*
Model 2	-0.01 (0.13)	-0.16 (0.06)	-0.33 (0.08)	0.049*

Mode 1 adjusted for age, sex, birth weight and maternal education
Model 2 adjusted for all covariates and food responsiveness
* P < 0.05

8.4 Discussion

This study provides the first evidence for an association between shorter nighttime sleep and higher food responsiveness, a measure of hedonic eating, in childhood. This study showed that higher food responsiveness could partly account for the linear relationship between sleep and BMI-SDS at age 5. In contrast, sleep duration showed no association with satiety responsiveness, a measure of homeostatic eating.

These findings support experimental work in adults which has suggested that acute sleep deprivation influences 'hedonic' rather than 'homeostatic' pathways to food consumption (Chaput & St-Onge, 2014; Markwald et al., 2013; evidence given in detail in Chapter 2, section 2.4). The results are also similar to the one small paediatric study which found that shorter sleep was associated with higher external eating, but not with emotional or restrained eating (Burt et al., 2013). External eating has conceptual overlap with food responsiveness in that both traits reflect the propensity to over-consume in response to palatable food cues.

Among adults, one previous study has shown that a tendency to disinhibited eating moderates the association between sleep and BMI, with a stronger relationship among adults who had higher disinhibited eating (Chaput et al., 2011). Disinhibited eating reflects the propensity to eat opportunistically within an obesogenic environment (Bryant, King, & Blundell, 2008), so again has similarities to the food responsiveness scale of the CEBQ. Furthermore, functional imaging studies have shown that sleep loss increases activation in reward centres of the brain, and subsequently food-desire, in response to images of highly palatable foods, but not in response to healthy food items (Greer et al., 2013). Together, these findings indicate that suboptimal nighttime sleep may specifically increase the salience of palatable foods, and consequently the drive to consume, within a permissive environment. Importantly, the present findings extend experimental work in adults to show that similar processes may be involved in very young children, and under free-living conditions.

Although this study provides evidence that sleep may increase hedonic eating in children, more work is needed to show that this in turn is what drives overconsumption among shorter sleepers. Studies on dietary intake have shown that shorter sleeping children have more frequent eating occasions and more unfavourable dietary patterns, in particular a higher intake of energy-dense foods (Golley et al., 2013; Hjorth et al., 2014; Kjeldsen et al., 2013). These patterns of consumption characterise a kind of 'hedonic overeating' (Yu et al., 2015), where eating is responsive to food cues rather than due to impaired satiety processes (Chaput & St-Onge, 2014).

There is also a need for longitudinal research to establish whether the relationship between sleep and eating behaviour strengthens as children gain increasing autonomy over their food choices and environment, and whether factors such as food availability, accessibility, and rules in the home influence susceptibility to weight gain. Given that parents largely control the food environment at a young age, this could have implications for interventions to prevent excess weight gain, for example by controlling exposure and access to palatable food items among children who experience difficulty sleeping. This may be particularly important at night when, as the results from study 3 suggest, parents may be more inclined to feed-to-soothe.

8.4.1 Limitations

This study has limitations that should be considered. Firstly, the cross-sectional design means longer-term follow-up is needed to understand the temporal distribution of the relationship between sleep, food responsiveness, and weight. Based on experimental evidence in adults, this study made the hypothesis that shorter sleep increases food responsiveness. However, the possibility that food responsiveness affects sleep cannot be excluded. For example, it could be that children who are more food responsive are also more difficult to settle at night when adults or older children are eating. This possibility should be explored in in future studies using a longitudinal design which includes multiple assessments of both sleep and food responsiveness.

The CEBQ was used to assess feeding behaviour styles. In keeping with previous reports (Wardle & Carnell, 2009) food responsiveness in this study was interpreted as indicating the involvement of hedonic processes. However, a careful inspection of the 5 questions that comprise the food responsiveness scale reveals the items may be considered to be related more to an appetite drive (e.g. my child is always asking for food) as opposed to a response to the pleasurable or satisfying aspects of foods. Accordingly, the interpretation is that an association with sleep duration may indicate an enhanced drive for food rather than an increased hedonic sensitivity.

The present sample included only children with complete data on sleep and eating behaviour, and for the mediation analyses, only a smaller sub-sample with BMI data; thereby excluding a considerable proportion of the Gemini sample. Children with complete data had mothers who were more likely to be university educated and who were more likely to be from a white ethnic background. This could limit the generalisability of the findings, so replication in a more diverse sample is required.

Gemini children are leaner with respect to UK 1990 reference values; reflecting the fact that twins tend to be born smaller than singletons (Buckler & Green, 2004). Although this could also limit the generalisability of the findings, there is no strong reason to expect that the association between sleep and weight, and the factors that mediate this relationship, should differ between twins and singletons.

As with each other study presented in this thesis, parent-reported nighttime sleep and BMI is a limitation, although this is common in larger population-based studies where objective measurements are not feasible. It is nonetheless encouraging that the mean nighttime sleep duration in this sample is comparable to published reference values for 5 year old children (see Figure 1.3, Blair et al., 2012). The limitations associated with parent-reported nighttime sleep are discussed in detail in the General Discussion.

8.4.2 Conclusion

This study shows that shorter sleep at age 5 is associated higher food responsiveness, an index of hedonic eating, but not with satiety responsiveness, an index of homeostatic eating. Mediation analysis is consistent with the idea that hedonic eating is part of the pathway mediating the effect of shorter sleep on adiposity.

Chapter 9 Study 5: The role of the home environment in the sleep-weight relationship

9.1 Background and aims

The findings presented in Study 4 suggest that changes in responsiveness to food stimuli (hedonic eating) may be part of the pathway through which insufficient sleep drives adiposity in childhood. Supporting this idea, studies of dietary intake in young children have demonstrated that shorter sleep is associated with patterns of consumption characterised by a greater intake of energy-dense food (see Chapter 2, section 2.2.2 for a review of the literature).

In early life the home food environment, which includes multiple physical and social aspects of food and feeding, is thought to exert a strong influence on consumption patterns and weight trajectories (Spurrier et al., 2008). For example, in the Gemini sample a higher risk home food environment, characterised by the greater availability and accessibility of food, as well as more permissive parent feeding practices, has previously been associated with poorer dietary behaviour at age 4 years. This included a lower intake of fruit and vegetables, and a higher intake of energy-dense snack foods, and sugar-sweetened beverages (Schrempft et al., 2015).

The notion that inadequate sleep may encourage eating that is responsive to the sensory properties of food (hedonic eating), raises the possibility that increasing exposure and access to food may make shorter sleeping children more susceptible to excess energy intake. However, no studies have examined whether the home food environment influences the susceptibility of children to weight gain with shorter nighttime sleep. This information could help to better understand the pathways through which shorter sleep may lead to overconsumption and weight gain in early life.

The aim of the present study was therefore to test whether the degree of risk within the home food environment moderated the cross-sectional association between sleep and weight observed at age 5. Given previous evidence linking sleep to food responsiveness

(Study 4), it was hypothesised that a stronger relationship between sleep and weight would be observed among children living in a higher risk home food environment.

9.2 Methods

9.2.1 Participants

Participants were a subsample of 390 families from the Gemini twin birth cohort, described in Chapter 4. Families were included if they had complete data on nighttime sleep duration and BMI-SDS at age 5, and had completed the home food environment interview. One child was randomly selected from each family to avoid clustering effects. Mothers who provided complete data were slightly older (34.5 versus 32.7 years, $P < 0.001$) and had a slightly lower BMI when the twins were born (24.4 versus 25.2, $P < 0.001$). Mothers who provided complete information were also more highly educated, and more likely to be from a white ethnic background (P's < 0.001).

9.2.2 Measures

9.2.2.1 Sleep

As with previous studies, nighttime sleep duration was calculated from parent-reported bedtime and wake time when the children were on average 5 years old. As in Study 4, sleep duration was categorized into 3 groups according to population means for nighttime sleep at this at this age (Blair et al., 2009): shorter (<11 hours a night), adequate (11-12 hours) and longer (>12 hours).

9.2.2.2 Home Food Environment

As reported in Chapter 4 (section 4.3.5), the home food environment was assessed via a computer-assisted telephone interview with the primary caregiver, a full transcript of which can be found in Appendix D. Briefly, the interview assessed multiple aspects of the food environment related to food availability, visual accessibility of food, physical accessibility of food, and parental feeding practices. A panel of 30 experts in childhood obesity were consulted, and variables relating to each domain were included in the composite if more than 60% of experts believed it to be associated either an increased or decreased risk of weight gain. In total 21 variables were combined to create the home environment risk score. Variables identified as being associated with a decreased risk of weight gain were reverse scored, then each variable was standardised and summed to create the composite score. In a random sample of 40 families, one-week test-re test reliability of the home food environment composite was high (0.71; 95% CI 0.52 – 0.83).

For the purposes of the stratified analyses presented in this study, the home environment score was dichotomised at the mean into high (above 0, the standardised mean) and low (below 0).

9.2.2.3 Body Mass Index Standard Deviation Score

As discussed in Chapter 4 (section 4.3.8) Gemini families are asked to provide 3-monthly height and weight measures using standardised equipment provided to all participating families (electronic weighing scales and height charts). Weight data included in the analysis were collected when the children were 5 years of age. As in study 4, if the parent had not provided 5-year weight data, data points available within 3 months of this age were used. Age and sex adjusted BMI-SDS were calculated using UK 1990 reference data (Cole, Freeman, & Preece, 1995).

9.2.2.4 Chaos

Chaos in the home was measured using a shortened version of the Confusion Hubbub and Order Scale (CHAOS; see section 4.3.6), a full copy of which is provided in Appendix E. This questionnaire assessed the general level of noise and disorder in the home using 6 items, for example, 'you can't hear yourself think in our home'. Items are responded to as true (1 point) or false (2 points) and summed to create a mean score, with higher scores indicating a greater level of chaos within the home. The CHAOS scale has been shown to correlate well with objective measures of home disorganisation and parenting, demonstrating good internal consistency (Cronbach's α= 0.79) and 12-month stability (r = 0.74) (Matheny, Wachs, Ludwig, & Phillips, 1995).

Home chaos is thought to reflect an underlying lack of structure in the home, and may be a marker of psychosocial stress in early life (Lumeng et al., 2014). Importantly, in previous studies chaos has been associated with higher food responsiveness, shorter sleep duration, and adiposity in young children (Appelhans et al., 2014; Lumeng et al., 2014). As chaos may influence the physical and social food environment as well as sleep patterns and eating behaviour, this factor was considered as a potential confounder in the adjusted analyses. It was also included as a covariate to exclude the possibility that any observed effects could be accounted for by an underlying lack of structure within the home.

9.2.2.5 Socio-demographic characteristics

As outlined in Chapter 4 (section 4.3.10) socio-demographic characteristics were recorded in baseline questionnaires. Mothers reported on their highest level of education, and for the purposes of the current study this was categorised into higher (university educated) middle (additional school or vocational qualifications), and lower (up to secondary schooling). Birth weight was measured by asking parents to copy or transcribe health records.

9.2.3 Statistical analysis

Pearson correlations were first run to examine the associations between chaos in the home, the food environment, nighttime sleep duration, and BMI-SDS at age 5 years.

Comparison of BMI-SDS between sleep groups was then carried out using Analysis of Variance (ANOVA) with polynomial contrasts testing for a linear association. ANOVA models were first run to examine the linear relationship between sleep duration and BMI-SDS. The sample was then stratified into two food environment risk groups by dividing the sample at the mean (higher, above the mean of 0; lower, below the mean). ANOVA models testing the relationship between sleep duration and BMI-SDS were run separately for children living in higher and lower risk home food environments. All models were repeated adjusting for the covariates age, sex, maternal education, birth weight and the CHAOS score using Analysis of Covariance (ANCOVA).

A stepwise multiple linear regression model was run to formally test the moderation effect. This model included nighttime sleep duration, the food environment composite, and a sleep-by-food environment interaction term predicting BMI-SDS. For the purposes of this model, all variables were entered as continuous. The adjusted model included the covariates age, sex, maternal education, birth weight, and the CHAOS score.

9.3 Results

Participant characteristics are given in **Table 9.1**. In total, 390 children with a mean age of 5.1 (SD 0.12) years were included in the analysis. The average nighttime sleep duration was 11.51 (SD 0.57) hours per night, and the average BMI-SDS was -0.20 (SD 1.1). The majority of the sample (55%) had a mother who was university educated.

Descriptive statistics for the home food environment by risk group (higher and lower) are shown in Table 9.2. Data are presented for each variable included in the composite score. Higher risk home food environments had a greater availability of energy-dense foods, and these foods were more physically and visually accessible to the children. The higher risk home food environments were also characterised by more permissive parent feeding practises (Table 9.2).

Pearson correlation coefficients for the association between the CHAOS score, the home environment risk score, nighttime sleep, and BMI-SDS are given in **Table 9.3**. Chaos in the home was significantly negatively correlated with nighttime sleep duration ($r = -0.17$, $P = 0.001$), and positively correlated with the home food environment risk score ($r = 0.17$, $P = 0.001$) and BMI-SDS ($r = 0.12$, $P = 0.02$), supporting its inclusion as a covariate in the adjusted analyses.

The results of the ANOVA and ANOCA models predicting BMI-SDS are given in **Table 9.4**. Shorter nighttime sleep was associated with higher BMI-SDS at age 5 (P for linear trend = 0.017). The linear association was retained after adjusting for age, sex, birth weight, maternal education, and the CHAOS score.

In total, 48.2% of children scored above the mean food environment risk score and were considered to live in a 'higher' risk home food environment. The stratified analysis demonstrated that there was a significant linear association between shorter sleep and higher BMI-SDS among children living in a higher risk home food environment (P for linear trend = 0.01), but that sleep was not associated with BMI-SDS among children living in a lower risk home (P for linear trend = 0.51). The linear associations were retained after adjusting for age, sex, birth weight, maternal education, and the CHAOS score (see **Table 9.4**).

To formally test for moderation, a stepwise multivariate linear regression model was run predicting BMI-SDS. In the unadjusted model, there was a significant main effect of sleep duration, such that shorter sleep was associated with higher BMI-SDS (β = -0.22; 95% CI: -.41 to -0.04, P = 0.02). However, the food environment risk score did not predict BMI-SDS in this model (β = 0.0; 95% CI: -0.02 to 0.0, P = 0.98). In addition, no significant interaction was observed between sleep duration and the food environment risk score in the prediction of BMI-SDS (β = 0.10 95% CI: -0.22 to 0.02, P = 0.11), indicating that the home food environment did not significantly moderate the linear relationship between sleep duration and BMI-SDS. These associations did not change in the multivariate model adjusting for age, sex, birth weight, maternal education, and the CHAOS score (Table 9.5).

Table 9.1. Participant characteristics given as mean (standard deviation) unless otherwise stated (n=390)

Age (years)	5.12 (0.12)
Sex (%) boy/girl	53.1 / 46.9
Maternal education (%) low/medium/high	24.4 / 20.8 / 54.9
Birth weight (kg)	2.48 (0.52)
CHAOS	0.39 (0.32)
Sleep Duration (hours)	11.51 (0.57)
BMI-SDS	-0.20 (1.05)

Table 9.2. Descriptive statistics for the home food environment variables. Data given as % (n) who responded 'Yes', unless stated otherwise.

Home food environment	Lower risk home (n=202)	Higher risk home (n=188)
Availability		
Number of fruit types, mean (SD)	8.70 (3.28)	7.13 (2.68)
Number of vegetable types, mean (SD)	11.70 (3.62)	9.95 (3.65)
Number of energy-dense snack types, mean (SD)	4.91 (2.09)	5.48 (1.93)
Presence of sugar-sweetened drinks	31.2 (63)	50.0 (94)
Accessibility (visibility)		
Fruit on display	97.0 (196)	92.0 (173)
Vegetables ready-to-eat	67.3 (136)	35.1 (66)
Energy-dense snacks on display	9.4 (19)	33.0 (62)
Sugar-sweetened drinks on display	3.0 (6)	11.2 (21)
Accessibility (child can help him/herself)		
Fruit	58.4 (118)	46.8 (88)

Vegetables	35.1 (71)	16.5 (31)
Energy-dense snacks	5.0 (10)	7.4 (14)
Sugar-sweetened drinks	1.0 (2)	3.2 (6)
Parental feeding practices, mean (SD)		
Emotional feeding (1 = never; 5 = always)	1.55 (0.50)	1.85 (0.55)
Instrumental feeding (1 = never; 5 = always)	2.14 (0.62)	2.50 (0.59)
Encouragement (1 = never; 5 = always)	2.08 (0.48)	1.71 (0.47)
Modelling (1 = never; 5 = always)	2.51 (0.58)	1.98 (0.66)
Monitoring (1 = never; 5 = always)	2.77 (0.84)	2.07 (0.77)
Covert restriction (1 = never; 5 = always)	3.34 (0.68)	2.72 (0.67)
Restriction (1 = not at all; 7 = strictly)	5.60 (0.89)	3.60 (1.65)
Family meal frequency (days per week)	4.33 (1.51)	3.60 (1.64)
Frequency child eats while watching TV (days per week)	0.96 (1.23)	1.43 (1.69)

Table 9.3. Pearson correlation coefficients for the association home environment variables, sleep duration and BMI-SDS

	Chaos	Sleep Duration	BMI-SDS	Home Environment
Chaos	1			
Sleep Duration	-0.17**	1		
BMI-SDS	0.12*	-0.13*	1	
Home food environment	0.17**	-0.15*	0.02	1

*P < 0.05, **P = 0.001

Table 9.4. Associations between sleep duration and BMI-SDS by home environment risk (higher and lower). Data given as mean, standard error.

Nighttime sleep	<11 hours	11-12 hours	>12 hours	P (linear trend)
Number	38	239	113	
BMI-SDS	0.06 (0.19)	-0.14 (0.06)	-0.41 (0.11)	0.017*
BMI-SDS[a]	0.05 (0.18)	-0.12 (0.07)	-0.42 (0.10)	0.024*
Higher risk food environment				
Number	25	115	48	
BMI-SDS	0.14 (0.26)	-0.12 (0.09)	-0.58 (0.21)	0.013*
BMI-SDS[a]	0.09 (0.25)	-0.07 (0.11)	-0.58 (0.18)	0.039*
Lower risk food environment				
Number	13	124	65	
BMI-SDS	-0.09 (0.28)	-0.15 (0.08)	-0.28 (0.11)	0.506
BMI-SDS[a]	-0.11 (0.24)	-0.14 (0.08)	-0.33 (0.11)	0.344

[a] Model adjusted for age, sex, birth weight, maternal education and chaos
*P < 0.05

Table 9.5 Linear regression analysis predating BMI-SDS at age 5. Main effects are given for sleep, the home environment risk score and the sleep x home environment interaction term

Unadjusted moderation model	**B**	**95% CI**	**P-value**
Sleep main effect	-0.22	-0.41 to -0.04	0.02*
HE main effect	0.00	-0.02 to 0.0	0.98
Sleep x HE	-0.10	-0.22 to 0.02	0.11
Adjusted moderation model[a]			
Sleep main effect	-0.22	-0.42 to -0.03	0.03*
HE main effect	0.00	-0.02 to 0.02	0.90
Sleep x HE	-0.08	-0.21 to 0.04	0.19

[a]Model adjusted for age, sex, birth weight, maternal education, and CHAOS
*P < 0.05

9.4 Discussion

This is the first study to show that living in a more permissive home food environment may strengthen the association between shorter sleep and higher weight in early life. Supporting the hypothesis, shorter nighttime sleep was associated with a higher weight among children living in a higher risk home food environment, but not among children living in a lower risk home food environment. This effect was observed independently of multiple confounding factors including chaos in the home, although replication in a larger sample is required to confirm the moderation effect.

Accumulating evidence (reviewed in Chapter 2, section 2.2 and 2.4) suggests that overconsumption is likely to drive weight gain during periods of short or insufficient sleep, and this may be caused by changes in hedonic eating. Supporting this idea, Study 4 demonstrated that shorter nighttime sleep was associated with higher responsiveness to food stimuli, and this could partly explain the relationship between sleep and weight at age 5. The results of the present study extend this work to show the inverse association between sleep and weight was observed only among children living in a higher risk home food environment. Importantly, palatable foods were more available and easily accessible within higher risk homes. This may suggest that without exposure to food stimuli, or the ability to access food either autonomously or via parental feeding practices, the eating behaviours that are hypothesised to be driving overconsumption may not be fully expressed. Similarly, being awake for longer will increase food cue exposure, and this could encourage overconsumption if the home food environment offers abundant cues to palatable food and enables additional eating opportunities. The results of this study therefore support the idea that hedonic overconsumption may be driving weight gain among shorter sleeping children (Study 4), but also demonstrate the potential importance of the home food environment in eliciting overconsumption. Importantly, the associations emerged independently of home chaos, suggesting that an underlying lack of structure within the home cannot account observed effects, and the findings are specific to physical and social factors occurring within the home food environment.

It is important to consider that multiple behavioural, biological and environmental pathways are likely to be involved in increasing the risk of weight gain among shorter sleeping children (Chaput & Tremblay, 2012). Although research to date has largely overlooked the home environment, understanding that the food environment may ameliorate the vulnerability of children to gain weight with shorter nighttime sleep could have important implications for intervention. Indeed, features of the physical and social home food environment may be more easily amenable to change than other factors thought to drive weight gain, such as food responsiveness (Study 4). Limiting the visual and physical accessibility of food cues within the home for example may be an important step to preventing excess energy intake. Given that previous work has shown additional energy intake is most likely to be consumed during the night (Study 3), controlling the food environment and feeding practices at night, when parents may be more prone to feed to soothe, may also be critical. However, more work is needed to clearly define the role of parental feeding practices in the sleep-weight relationship.

Although there was evidence for moderation in the stratified analyses, no significant interaction between sleep duration and the food environment was observed in the linear models. However, the confidence interval only marginally passed zero, indicating the null result may be an issue of power due to the small sample size ($n = 390$). Indeed, a post-hoc power calculation indicated that to have sufficient power (95%) to detect the interaction effect ($b = -0.08$) a sample of 2,020 would be required. Therefore, the study requires replication in a larger sample before the moderation effect can be confirmed. Nevertheless, it remains important to identify factors that may alter the association between sleep and weight. Not only can this can help to understand the mechanisms underlying this sleep-weight relationship, but it may also explain why in population-based studies the effect-size for the simple relationship is generally small, leading some researchers to question its clinical significance (Magee, Iverson, Huang, & Caputi, 2008).

9.4.1 Strengths and limitations

There are limitations in this study that should be acknowledged. Firstly, data were included from families who had provided complete information on sleep, the home food environment and BMI-SDS at age 5 years (n = 390). There has been some degree of attrition over the 5-year study period, so this excluded a substantial proportion of the baseline Gemini sample (74%). As discussed, the smaller sample size may mean the study was underpowered to detect the interaction effect.

There were also some differences between the study sample and those families who did not provide complete data. Specifically, mothers in this sample were more likely to be university educated and were more likely to be from a white ethnic background. Therefore, caution should be taken when generalising the findings to other populations. It would be beneficial to replicate this study in a larger socio-demographically diverse sample.

Twins tend to be born smaller than singletons, and remain smaller through early life (Buckler & Green, 2004). Reflecting this, the sample analysed were leaner with respect to 1990 reference values for age- and sex- standardised BMI scores (mean BMI-SDS = -0.20). Although this could also limit the generalisability of the findings, there is no strong reason to expect that the association between sleep and weight, and the factors that moderate this relationship, should differ between twins and singletons.

Parent reported nighttime sleep is an important limitation, which is discussed in detail in the General Discussion. However, it is reassuring that the nighttime sleep duration reported in this sample (11.5 hours per night) is comparable to published reference values for singleton children aged 5 years (11.3 hours, Figure 1.3) (Blair et al., 2012). There was also a small time lag between measurement of the food environment (4.2 years) and the sleep and weight measures (5 years). However, population means suggest nighttime sleep duration is relatively similar at these two ages (Blair et al., 2012). It would be preferable however for future studies to collect multiple concurrent measures.

A key strength of this study is the use of a composite measure that considered both physical and social aspects of the home food environment, albeit that they were self-reported. Indeed, multiple factors with the home environment are known to exert small effects on weight, so composite measures provide an important means to assess overall risk to adiposity (Schrempft et al., 2015). Furthermore, the composite indicator used in this study was based on guidance from an expert panel, included multiple physical and social features of the home food environment, and demonstrated moderate test-retest reliability (see Chapter 4, section 4.3.5). Although the composite score was not associated with BMI-SDS in this study, it has previously been associated with energy balance behaviours in the Gemini sample, so it may be that associations with weight are not observed until a later age (Schrempft et al., 2015).

9.4.2 Conclusion

The association between shorter sleep and weight is stronger among children living in a more permissive home food environment, suggesting the food environment may facilitate overconsumption among shorter sleeping children. The home environment offers a potentially important target for intervention to prevent excess weight gain among vulnerable children.

Chapter 10 General Discussion

10.1 Prelude

The overall aim of this thesis was to understand sleep behaviour in early life, and to identify the behavioural pathways through which it may contribute to the development of adiposity. In order to achieve this I completed 5 studies, which together have helped to develop an understanding of sleep and its relationship to eating behaviour and weight in the first 5 years of life. I would argue that the findings presented in this thesis provide a strong argument for the idea that energy intake drives weight gain among shorter sleeping children. These findings have important implications for interventions, and highlight opportunities for future work in this emerging area of obesity research. In this chapter, the thesis findings, implications for practice, strengths and imitations, and avenues for future research are presented and discussed.

10.2 Summary of the thesis findings

There currently exists a body of research examining the environmental and socio-demographic predictors of shorter nighttime sleep in children; however identifying the predictors and pathways to shorter nighttime sleep in a large sample of young children was novel (Study 1, Chapter 5). In Study 1, many well-established environmental (TV viewing) and socio-demographic (ethnicity, maternal education) correlates of shorter nighttime sleep were found to operate through a later bedtime. This is an important finding as it provides a clear target for intervention (bedtime) for otherwise stable but key influences on sleep behaviour (ethnicity, maternal education). Bedtime is already emphasised in sleep hygiene recommendations (Mindell, Meltzer, Carskadon, & Chervin, 2009), but these findings suggest some groups may need a specific emphasis on the importance of an early and consistent bedtime routine. Indeed, identifying that children who go to bed later at night do not tend to fully compensate by waking later in the morning seems crucial, and suggests an early bedtime may be an essential component to healthy sleep practices in young children, and a necessary step to achieving adequate nighttime sleep. Demands on morning wake time will become increasingly prominent as the child reaches school age and must adhere to school start times, meaning the ability to compensate for sleep lost at night by sleeping later in the morning will become less likely through older childhood and adolescence. This is tantamount to the idea of 'social jetlag' in adults which refers to sleep loss caused by the discrepancy between the internal versus social clock (Parsons et al., 2014), and which may be a key contributor to declining nighttime sleep among westernised adult populations (Knutson, Van Cauter, Rathouz, DeLeire, & Lauderdale, 2010). Indeed, there is some evidence that sleep may track in early life (Taveras et al., 2014), so identifying the predictors and pathways at a young age provides an important contribution to the existing paediatric sleep literature.

Understanding the predictors and pathways to shorter sleep in young children was important not only because sleep may track in early life, but also because insufficient sleep is a key contributor to health and wellbeing, and is a recognised a risk factor for adiposity in young children (Cappuccio et al., 2008). In order to explore what could be driving the association between shorter sleep and adiposity, Study 2 (Chapter 6) examined the relationship between parent-reported nighttime sleep and energy intake, which was assessed using 3-day diet diaries at age 21 months. At this age, children sleeping <10 hours a night were found to consume on average 100 kcal more each day than children sleeping ≥13 hours a night, with a linear association in between. Although these differences in energy intake may seem small, 100 kcal accounts for approximately 10% of total daily energy intake at this age. Indeed, if sustained over the longer term, small differences in energy intake have the potential to shift the distribution of overweight and obesity (Boyd Swinburn et al., 2011). Importantly, these findings are in line with many experimental studies of sleep restriction in older children and adults (Hart, Cairns, & Jelalian, 2011; Markwald et al., 2013), but this study provided the first evidence in a population-based sample of young children. Using the dietary data it was also possible to show that grams per day of carbohydrate and fat were higher among shorter sleeping children, but that the relative differences were small, and when considered as a proportion of total energy there were no differences in the composition of the dietary intake between the 5 sleep groups. This finding suggests that shorter sleeping children were not consuming different diets at this age, but were simply consuming more. The results implicate excess energy intake as part of the pathway through which shorter sleep impacts adiposity in early life. Moreover, that the associations were observed before a relationship with weight had emerged strengthens the argument for a causal role, but of course cannot confirm causality.

Identifying an inverse association between sleep and energy intake made an important contribution to the paediatric sleep and weight literature. However, this work did not help to identify why shorter sleeping children were consuming more. Study 3 (Chapter 7) used the diet diary data from 21 months to further explore when and how additional calories were being consumed among shorter sleeping children. Specifically, differences in energy intake were tested by time of day (morning, afternoon, evening, night) and by eating occasion (meal, snack, drinks) across the 5 sleep groups. The results showed that shorter sleeping children consumed more energy during the nighttime period only, and that these calories were predominantly consumed from milk drinks before midnight. There were no differences in energy intake by meal or snack occasions. This was the first study to examine the temporal distribution of energy intake by nighttime sleep duration in young children. The finding that additional energy was consumed at night is in line with experimental literature, which has shown that individuals experiencing enforced sleep restriction tend to consume excess calories at night, and often from snack-based food (Nedeltcheva et al., 2009). Demonstrating that shorter sleeping children consumed more energy from milk drinks is novel, and I believe has important implications for interventions and for future research (these are discussed further in this chapter under sections 10.3 and 10.5). Although parents were not asked why they were feeding at night, providing milk drinks is a common strategy used by caregivers to soothe children before bed (Mindell, Sadeh, Kohyama, & How, 2010). However, providing additional calories at night and from milk drinks is concerning for a number of reasons. Firstly, milk is high in protein, and excess protein intake before the age of 2 years may adversely impact adiposity risk (Michaelsen & Greer, 2014). Furthermore, regularly providing milk before bed may lead to this behaviour becoming habitual, and could therefore operate to dissociate feeding from hunger (Lally & Gardner, 2013). Consuming food at night has also been shown to be detrimental to health, independent of total energy intake (Baron et al., 2011b; Morgan, Shi, Hampton, & Frost, 2012). Therefore, parents of children with more problematic sleep behaviour should be strongly encouraged to use alternative methods to soothe at night as part of the bedtime routine. Although the consumption of milk drinks at night may be specific to children of this age, it is the forming of habits among shorter sleeping children (eating at night or eating to self-soothe) that may be most critical. As children get older and gain greater independence over their eating behaviour, it may be that milk drinks are simply replaced by energy-dense snack foods. Study 1 highlighted the importance of an early bedtime, and demonstrating that shorter sleeping children consume additional calories at night (after the mean bedtime, but before midnight) further underscores the significance of this practice for young children.

The results from Studies 2 and 3 suggest that excess energy intake may lie on the pathway between insufficient sleep and weight gain in children. Although individual level factors will contribute to excess energy intake at this age, parents largely determine what foods young children eat and when, and so must be partly responsible for milk feeding at night. Just as sleep-related differences in adiposity may take time to emerge, differences in eating behaviour between shorter and longer sleepers may only express themselves when the child has a given level of autonomy over their feeding behaviour. In addition to dietary intake, it is important to understand how habitual sleep impacts patterns of eating behaviour in young children, as this information could help to better understand the behavioural pathways through which shorter sleep impacts adiposity risk at a young age. This is particularly pertinent in light of emerging research in adults suggesting that insufficient sleep predominantly impacts hedonic (reward driven) as opposed to homeostatic (driven to maintain energy balance) pathways underlying the drive to consume (Chaput & St-Onge, 2014).

To explore these ideas further, Study 4 examined the relationship between nighttime sleep at 5 years and eating behaviour phenotypes known to be associated with adiposity in childhood – food responsiveness and satiety responsiveness (from the Child Eating Behaviour Questionnaire; Wardle et al., 2001). Food responsiveness is the tendency to want to eat in response to externally perceived food stimuli so reflects a kind of hedonic eating, while satiety responsiveness is the tendency to stop eating based on internal cues to fullness so reflects a kind of homeostatic eating (Wardle et al., 2001). Given previous research in adults demonstrating acute sleep deprivation increases responsiveness to palatable foods (Greer et al., 2013), the objective of Study 4 was to test the hypothesis that shorter sleeping children are more food responsive, and to explore the mediation of the relationship between sleep and weight by food responsiveness (hedonic eating). Importantly, Study 4 found a significant linear association between shorter sleep and higher food responsiveness at age 5. In a subset of the sample with BMI data, food responsiveness was found to significantly mediate the inverse association between sleep and weight at this age. However, sleep was not associated with satiety responsiveness, suggesting that shorter sleep in early life may specifically impact hedonic as opposed to homeostatic pathways to food consumption. These findings support recent neuroimaging data in adults (Greer et al., 2013; St-Onge et al., 2012), but this study provided the first evidence that sleep may impact hedonic eating in children under free-living conditions. Although this study did not include a measure of dietary intake (dietary data were not collected at 5 years), the findings are also consistent with studies in older children showing that short sleep is associated with poorer dietary habits and a higher intake of energy dense foods (Hjorth et al., 2014; Kjeldsen et al., 2013). This study made a significant contribution to the literature by identifying a clear behavioural pathway through which shorter sleep may impact energy intake and weight gain in early childhood.

For children living in more 'obesogenic' environments, alterations in food responsiveness could favour excess energy intake and weight gain. If increased food responsiveness can partly explain why shorter sleeping children are more prone to adiposity as the results of study 4 suggest, then living in an environment with abundant palatable food may make shorter sleeping children particularly susceptible to weight gain. Study 5 tested this hypothesis by examining whether the home food environment moderated the sleep-weight association observed at age 5. Supporting the hypothesis, a significant inverse association between sleep and weight was observed among children living in higher risk home food environments, but not among children living in lower risk environments. However, the interaction effect was not significant in the final model, possibility due to issues of statistical power (discussed further in section 10.4.2.6 of this chapter). Nonetheless, this study was important in that it identified the potential role of the home food environment in eliciting overconsumption among shorter sleeping children, thereby helping to better define the behavioural pathways through shorter sleep may lead to weight gain in early life.

The home food environment is known to be associated with energy balance behaviours in early childhood (Schrempft et al., 2015), but this was the first study to examine how it may impact the sleep-weight relationship in early life. Defining the role of the home food environment is important as it may be more easily amenable to intervention than other factors thought to drive overconsumption such as food responsiveness.

10.3 Implications for practice

The findings presented in this thesis could have important practical implications for interventions to improve child sleep and prevent excess weight gain in early life. The significance of obtaining adequate sleep, and of establishing healthy sleep habits in early life is well known (Carvalho Bos et al., 2009; Cespedes et al., 2014).The findings from Study 1 suggest that an early bedtime may be one of the most important influences on nighttime sleep. The results also indicate that adopting a bedtime routine that does not include exposure to television may be crucial; although this could be extended to other screen-based media (Chahal, Fung, Kuhle, & Veugelers, 2013; Shenghui et al., 2007). An early bedtime is already highlighted in many sleep hygiene recommendations (Mindell et al., 2009), and The American Academy of Paediatrics (AAP) recommends that parents and caregivers minimise or eliminate media exposure for children under the age of 2 years. However, interventions may be required to raise awareness and increase caregiver knowledge, particularly in lower socio-economic and ethnic minority groups. The findings from Study 1 suggests that children from these groups get less sleep at night because they go to bed later, and do not tend to compensate by waking later in the morning. I am aware of one behavioural intervention that targeted the bedtime practices of infants and toddlers. In this study, mothers were instructed to adopt a 3-step bedtime routine involving a bath, a massage, and a quiet activity (e.g. cuddling or reading a book) (Mindell, Telofski, Wiegand, & Kurtz, 2009). After 3 weeks, significant improvements were observed in night wakings and sleep latency, but not in sleep duration. However, the intervention did not specifically target the timing of the bedtime routine. While it is encouraging that a simple intervention can lead to significant improvements in infant and toddler sleep, there is clearly scope for more intervention work in this area.

It may also be important to consider that many parents and caregivers will use milk feeding at night as part of the bedtime routine, possibly as a means to soothe their children before bed (Mindell, Sadeh, Kohyama, et al., 2010). However, the findings from Study 3 suggest that milk consumption at night contributes to shorter sleeping children consuming more, and could therefore increase the risk of overweight. In addition to raising awareness around an early bedtime, parents and caregivers need to be encouraged to use alternative strategies to soothe their children before bed. For example, incorporating a warm bath or story-telling might provide useful substitutes for television viewing or milk feeding at night, the benefits of which have already been reported (Mindell, Telofski, Wiegand, & Kurtz, 2009).

Intervention strategies aimed at minimising milk feeding at night may also be important to consider in light of the high protein content of milk (3.4 grams per 100 grams in semi-skimmed), and recent evidence linking protein intake with excess weight gain in early life (Michaelsen & Greer, 2014). Interestingly, many baby nutrition companies have developed follow-on formula feeds specifically for use at night, as part of a bedtime routine. For example, *HiPP Organic* markets a product called 'Good Night Milk', which it advertises on the packaging as being 'an ideal complement to your baby's bedtime routine – from 6 months onwards'. Even the NHS website offers the following advice for children that wake up during the night,

> "*Is it hunger? If your child is a year or older, some cereal and milk last thing at night might help them sleep through the night*." (http://www.nhs.uk/Conditions/pregnancy-and-baby/Pages/sleep-problems-in-children.aspx#close).

However after weaning, milk at night should no longer be necessarily (Touchette et al., 2005a), and doing so routinely may operate to dissociate feeding from hunger, which could lead to overconsumption. This is also a concern for future weight gain, particularly when the child gains the autonomy to make decisions concerning when and how much to consume.

By age 5, the findings from Study 4 suggest that shorter sleep may increase responsiveness to food stimuli, and that this may be part of the pathway through which sleep influences weight gain. Furthermore, children living in more obesogenic home food environments may be particularly vulnerable to the effects of shorter nighttime sleep (Study 5), probably because these environments offer abundant cues to food, that are also more easily accessible. More work is required to understand whether improving sleep could lead to reductions in food responsiveness. This phenotype has been shown to be highly heritable and is strongly associated with adiposity in early life (Llewellyn, Jaarsveld, Johnson, Carnell, & Wardle, 2010), so this finding could have meaningful implications for interventions to prevent overconsumption in childhood. With regards to the relationship between sleep and weight, the findings indicate that it is not a failure of satiety, but an increased responsiveness to food that may be driving the weight gain associated with shorter sleep, and that differences in the environment may impact the expression of this phenotype. Crucially, more food responsive children will not be able to eat opportunistically if their immediate environment does not offer the eating opportunities to do so. Parents largely control the food environment at this stage, so intervention strategies aimed at making the home environment less 'obesogenic' could be crucial. For example, parents could be advised to remove cues to palatable food in the home and reduce their accessibility (either physically or via changes in parental feeding styles). Replacing palatable food with healthy snack items (fruits and vegetables) might be another option. Of course, as children grow older they gain the autonomy to make decisions about when and how much to eat, and also spend less time at home, so controlling the environment will become increasingly challenging.

To summarise, the findings presented in this thesis provide clear points for exploration that could help to inform interventions. Before 2 years of age, establishing an early and consistent bedtime routine, discouraging nighttime use of screen-based media, and establishing soothing bedtime practices that do not involved milk (or other) feeding could help to encourage healthy sleep and reduce the risk of overconsumption in early life. By age 5, reducing exposure to palatable food, as well as their physical accessibility, might provide a useful strategy to prevent overconsumption among sorter sleeping children.

10.4 Strengths and limitations

The findings presented in this thesis could have important practical implications. However, there are also a number of important strengths and limitations that should be acknowledged.

10.4.1 Strengths

A key strength of the Gemini cohort is that multiple measures of sleep, eating behaviour and weight have been collected in a large population-based sample of children from the first few months of life. This has allowed for the relationship between sleep, eating behaviour and weight to be examined from very early in life (15 months), and prior to any observed relationship between sleep and weight (Study 2, Chapter 6 and Study 3, Chapter 7). The large sample size has allowed for small associations with weight and sleep to be estimated, and the multiple assessments of weight, and factors relating to weight in early life, has meant that a number of potentially important covariates could be included in the analyses.

The dietary assessment at age 21 months reflects a key strength of the Gemini data (Studies 2 and 3). This is the largest dietary data set of its kind for toddlers in the UK, and the method has been validated for the assessment of dietary intake in children up to 24 months (Lanigan et al., 2001). The detail included in the diaries allowed for a comprehensive analysis of where and when additional calories were being consumed among shorter sleeping children (Study 2). This would not have been possible with other methods of dietary assessment frequently employed within the literature (for example, a food frequency questionnaire). Furthermore, studying the associations between sleep and dietary intake from a young age, before the emergence of an association with weight, supports, although does not confirm, a causal role for energy intake.

Another unique strength was the use of a comprehensive measure of the home food environment (Study 5). The home food environment includes multiple physical and social aspects of food and feeding, each of which are hypothesised to have a small effect on weight (Schrempft et al., 2015). Different facets of the home may operate in conjunction or oppose one another, so a composite measure has advantages over investigating single features (Schrempft et al., 2015). The composite risk score used in Study 5 was developed with the guidance of a panel of 30 experts in childhood obesity. The measure allowed Study 5 (Chapter 9) to examine the moderating role of the food environment in the sleep-weight relationship. The home environment has largely been ignored in the literature to date, but could provide an important avenue for intervention.

10.4.2 Limitations

Despite the strengths of the studies presented in this thesis, there are also a number of important limitations.

10.4.2.1 Sleep assessment

The reliance on parent report to measure nighttime sleep reflects a key limitation of the work presented in this thesis. Although the Gemini team applied for a BBSRC grant to obtain objective measures of sleep, this grant application was unfortunately unsuccessful. Some evidence has shown that, relative to objectively measured sleep (actigraphy), parents report that their children fall asleep earlier, and also tend to overestimate the sleep period by approximately 30 minutes (Kushnir et al., 2013; Nelson et al., 2014). However, studies have also shown good correspondence between parent-reported nighttime sleep and objective measures (Kushnir et al., 2013; Avi Sadeh, 2004). For example, in a sample of preschool children aged 4-6 years, the correlation between actigraphy and parent reported nighttime sleep was $r = 0.85$, $P < 0.0001$ (Kushnir et al., 2013). Parent report might also provide a better global representation of sleep behaviour than a few nights of objective recording. In a subsample of 40 Gemini families, 1 week test-retest reliability for sleep duration at age 4 years was excellent (intraclass correlation 0.89; 95% confidence interval 0.76– 0.95 for sleep duration). It is also encouraging that at each age studied, the mean sleep duration reported in the Gemini sample was comparable to published reference values for children of that age (Blair et al., 2012; Iglowstein et al., 2003). Although the utility of parent report may be acceptable in large population-based studies, it will be necessary to replicate the findings presented here using objective measures of nighttime sleep in smaller samples. Indeed, by reducing measurement error, objective measures should strengthen the effect size for the observed relationships between sleep, eating behaviour and weight in early life.

Although the correspondence between parent report and actigraphy measured sleep duration is generally high, low correlations have been observed for parent estimated wake after sleep onset and night waking (Kushnir et al., 2013). Ideally, total sleep time should be calculated as the time elapsed between the first period of sleep and the final awakening, with time spent awake at night deducted. Given that parents are less accurate at reporting other parameters of nighttime sleep, and in particular night-waking, the studies in this thesis calculated nighttime sleep as the time elapsed between parent-reported bedtime and waketime. It follows that reliance on parental report also meant it was not possible to examine other aspects of sleep in early life. This is a limitation given that there is some evidence to suggest that other factors including sleep timing, sleep quality, sleep variability, and sleep compensation may also impact eating behaviour and weight gain (Gonnissen, Hursel, Rutters, Martens, & Westerterp-Plantenga, 2013; Gonnissen, Mazuy, et al., 2013; Rutters et al., 2012). It is important to consider that the findings presented here only relate to parent reported nighttime sleep, and therefore do not extend to other potentially important dimensions of sleep. However, this is a key avenue for future research, discussed further in section 10.5.2 of this chapter.

Regardless of how it is measured, a general limitation with studying the impact of short sleep in early life is that it is not yet clear what constitutes 'optimal' nighttime sleep. Although population means can provide some indication of what is 'typical' for a given age, these are nonetheless time and population specific. What is 'optimal' will depend on the individual, their sleep needs, as well as previous sleep debt (Galland, Taylor, Elder, & Herbison, 2012). Without a clear indication of what is optimal, sleep was categorised into groups throughout this thesis, and linear trends between the groups were examined in studies 2, 3, 4, and 5. Among children under the age of 5 years there is good evidence to suggest that sleeping <11 hours a night is associated with an increased risk of overweight and obesity (Chen et al., 2014), and this was used as a justification for the point at which sleep duration was dichotomised in Study 1.

Categorising sleep also had the benefit of allowing comparisons to be made with the sleep-weight literature, which has generally used a variety of cut-points to define 'short sleep'. It also allowed for linear trends to be examined in the data which helps to strengthen the argument for a causal role between sleep duration and the outcome variable in each study chapter (Mann, 2003). It is important to note that for studies investigating eating behaviour (Studies 2, 3 and 4) the shortest and longest sleeping groups were often compared. However, given that evidence suggests sleeping <11 hours a night significantly increases a child's risk of adiposity (Chen et al., 2008), comparisons with this group for differences in energy intake and eating behaviour may be more useful.

10.4.2.2 Measurement of adiposity

The reliance on parent-reported height and weight also reflects a key limitation with the Gemini data. However, to minimise measurement error, all families were provided with standardised electronic weighing scales and height charts (replaced on request), alongside detailed instructions on how to accurately weight and measure their twins. There is evidence to show that parents can accurately weigh and measure their twins at home, when provided with standardised equipment (Himes, 2009). Furthermore, the height and weight data included used in this thesis were extensively cleaned by a trained statistician, with growth trajectories examined for each child.

There are limitations associated with using BMI-SDS as measure of adiposity more generally. While BMI is convenient for use on population-based studies, it is a measure of excess weight (corrected for height) rather than a measure of excess fat, and therefore does not distinguish between fat mass and fat-free mass. BMI corresponds well with objective measures of body fatness in adults (Deurenberg et al., 2001), but the association appears more variable in children due to developmental changes in fat mass (Freedman & Sherry, 2009). There is some evidence that BMI-SDS is a better indicator of adiposity among relatively fat children, but among relatively thinner children, differences in BMI can be accounted for by fat-free mass (Freedman & Sherry, 2009). Given that twins tend to be born smaller, and are leaner than singletons (Buckler & Green, 2004), this may be an important issue to consider when using BMI data from a twin sample. Parent-measured waist circumference could have provided additional information on abdominal adiposity, but this data was not collected in the Gemini sample.

Obtaining objective measurements of adiposity would have been preferable, but there was no funding available to collect this data in the Gemini sample. However, by reducing measurement error, objective measures should in theory strengthen the observed relationships between sleep, eating behaviour and adiposity. Indeed, using objective assessments that distinguish between fat mass and fat-free mass will be essential for future work in this area. There is some evidence to suggest that sleep could preferentially impact abdominal adiposity in children (Chaput & Tremblay, 2007), so methods such as dual x-ray absorptiometry (DXA) should be used to examine whether sleep impacts the deposition of fat in the body. This work could specifically help to better understand the relationship between shorter sleep and metabolic risk in early life (Cespedes et al., 2014).

10.4.2.3 Cross-sectional design

All of the studies in this thesis used cross-sectional data, which means it was not possible to make conclusions about causation or indeed the temporal distribution of any of the hypothesised relationships. The possibility of reverse causation can therefore not be excluded. Although there was a slight time lag between the sleep (16 months) and dietary (21 months) assessments included in Studies 2 and 3, this period of time was not long enough to be considered longitudinal. Issues of reverse causation may indeed be particularly important to consider in Studies 2, 3, and 4. For example, it is possible that daily energy intake or energy intake at night affected subsequent sleep initiation and consolidation. To my knowledge no studies have directly examined the impact of milk feeding at night on subsequent sleep duration, but sleep and feeding disturbances do tend to coexist in early life, so this is a possibility (Tauman et al., 2011). In Study 4, it is possible that more food responsive children are also more responsive to external stimuli within their environment, which may make them difficult to settle at night, although I am not aware of any studies that have specifically examined this. Equally, children who are more responsive to food could be more easily stimulated in the evening when older children and adults may be eating. Therefore, longitudinal studies including multiple measures of both sleep and eating behaviour will be necessary to disentangle the effects and clarify the temporal distribution of the relationships explored in this thesis. A second wave of data collection is currently underway in Gemini, collecting 3-day dietary records and sleep (via parent report) concurrently at age 7 years, but data are currently only available for 100 families.

The cross-sectional design also means the possibility that the relationships presented in this thesis are due to extraneous variables cannot be excluded. While each study attempted to control for potentially confounding factors, it is possible that there were other explanatory factors that were not measured and so could not be included. Indeed, there are a number of behavioural, health and environmental variables that could potentially confound the relationship between sleep, eating behaviour and weight in young children. Psychological and chronic illness, alcohol consumption and smoking are often cited as major factors that may confound the sleep-weight relationship in adults (Magee et al., 2008), but these factors are largely irrelevant in a sample this young. However in children, I would argue the omission of any reasonable measure of physical activity reflects a significant limitation of this thesis. Although studies investigating the relationship between sleep duration and physical activity have produced mixed results (Chapter 2, section 2.2.1), it remains that physical activity could meaningfully impact sleep duration, eating behaviour, and weight in young children. Energy balance is a dynamic homeostatic process, and clearly defining the role of physical activity in the relationship between sleep, eating behaviour, and weight in early life will be a necessary progression in the field. However, Gemini was initially set up with a focus on eating behaviour and growth, and although the Gemini team subsequently applied for funding to collect both objective sleep and physical activity data (accelerometer) from the BBSC, this grant application was unfortunately unsuccessful. This remains a key area for future research.

Where possible throughout the thesis I have attempted to draw parallels with existing experimental literature in both children and adults. This is because an experimental study which examines the impact of sleep restriction or extension on a given variable (for example, eating behaviour) provides the best possible paradigm to delineate cause and effect. Given the limitations involved in conducting cross-sectional observational research, highlighting that the findings are in line with experimental work strengthens the argument for a causal role. Although no experimental studies of sleep restriction or extension have been conducted in very young children, the possibility of reverse causation or confounding by a third variable becomes less likely when the results are considered alongside existing experimental work.

10.4.2.4 Generalisation to singletons

In early life, twins differ from singletons on a number of characteristics, particularly related to growth. Twins tend to be born 3-4 weeks earlier than singleton children (Phillips, 1993), have a lower birth weight, and consequently experience a period of rapid 'catch-up' growth in early life (Naeye, Benirschke, Hagstrom, & Marcus, 1966). These differences are reflected in the Gemini sample; the Gemini twins are leaner with respect to UK 1990 reference values generated from singleton children (Cole et al., 1995). Indeed, at age 5 years only 46 children (5%) were classified as overweight and only 9 (1%) as obese (Chapter 4, Table 4.8). This limited upper-BMI range meant it was not possible to examine associations with overweight or obesity more specifically.

In reference to sleep duration, it is conceivable that twins may keep each other awake at night. Approximately 82% of all families surveyed in the home environment interview (n = 1,113) reported that their twin twins currently shared a bedroom. Nevertheless, it is encouraging that the mean nighttime sleep in this sample is comparable to singleton children of a similar age (shown in Chapter 1, Figure 1.3) (Blair et al., 2012). Furthermore, the findings presented here are generally in line with studies of singleton children investigating similar relationships (Burt et al., 2013; Hoppe et al., 2013). In populations of singleton children, the wider variation in weight and larger percentage of children who are either overweight or obese may also mean that the observed associations between sleep, eating behaviour and weight are stronger, but this warrants further investigation.

Issues of generalisability associated with using data from a twin sample should be acknowledged, and the findings replicated in samples of singleton children. However, I am not aware of any strong reason as to why the predictors of shorter sleep or the associations between sleep, weight and eating behaviour should differ between twins and singletons.

10.4.2.5 Generalisation to other populations

Gemini is a population-based cohort, approximately representative of the UK population. However, as with other population-based cohorts, ethnic minority and lower socioeconomic groups are underrepresented. Attrition throughout the 5 year study period has also meant that socioeconomic and ethnic differences between responders and non-responders have become more pronounced. Consequently, the present work needs to be replicated in more diverse samples, and caution should be taken in generalising findings in this thesis to ethnic minority and lower socioeconomic groups. This is particularly important to consider given that ethnicity and socioeconomic status are known to be strong determinants of both sleep duration and weight in early life (Mindell et al., 2013; Mindell, Sadeh, Wiegand, et al., 2010; Reilly et al., 2005). For example, children from lower socioeconomic and ethnic minority families tend to go to bed later, and have less interactive hygiene-related bedtime routines (Milan et al., 2007). It is plausible then that feeding practices at night may differ among these groups, as may sleep related changes in eating behaviour. Although research with these groups is limited, it is encouraging that studies investigating the association between sleep and weight in lower socioeconomic groups have tended to find consistent results to those examining predominantly white, higher socioeconomic samples, such as Gemini (Miller et al., 2014).

10.4.2.6 Power

The Gemini study is large population-based cohort; however power may have been a limitation in Study 5 (Chapter 9). This study included only 16% (n = 390) of the baseline Gemini sample. The smaller sample size can be attributed to attrition in the Gemini study over the 5-year study period, but also to the number of variables included in this study from different assessment points (the home environment interview, the 5 year questionnaire, and weight and height data). The smaller sample size may have meant that Study 5 was not adequately powered to detect the moderation effect of the home food environment. Indeed a post-hoc power calculation, indicated that to have sufficient power (95%) to detect the interaction effect (b = -0.08) a sample of 2020 would be required. Replication in a larger sample would therefore be beneficial.

Including both twins in each analysis and adjusting for clustering using complex samples analysis would have increased the sample size of each study. However, the high intra-class correlations between twins for sleep, energy intake, and eating behaviour meant that doing so over-adjusted the data. In discussions with a statistician, it was decided that a random selection of 1 twin per family was the best approach to use when analysing the data.

10.5 Avenues for future research

Although there are limitations with the studies presented in this thesis, the findings have important implications for future research. Some implications were addressed alongside the limitations; in particular, there is a need for longitudinal studies with objective measures of sleep, adiposity, and physical activity. However, the scope for future research in this area is broad; some ideas are discussed below.

10.5.1 Sleep extension and randomised control trials

While studies of controlled sleep restriction have been pivotal in understanding the mechanisms by which sleep might generate weight gain, there is now a need for prospective, randomised controlled trials to examine the impact of sleep extension on eating behaviour and body weight. Conducting such studies among overweight and obese individuals who experience chronic short sleep will be particularly important, as this work will help to understand the utility of sleep extension in the treatment of overweight and obesity.

At present, one prospective randomised controlled trial is currently underway in the United States (Cizza et al., 2010). This trial is using non-pharmacological behaviour-based interventions to increase sleep duration to approximately 7.5 hours a night in habitually short-sleeping obese adults (sleeping <6 hours per night at screening). Anthropometric, endocrine, metabolic and psychological effects of sleep extension are being monitored over a 4-year study period. Interim findings suggest the sleep intervention has been successful, with participants in the intervention arm reporting improved mood and energy, more willingness to exercise, and diminished cravings for sweet or salty snacks in the evening (Cizza et al., 2010). It would be beneficial to examine similar effects in childhood.

Another way to assess the utility of sleep extension without running costly and time consuming randomised trials is to examine the impact of spontaneous changes in sleep duration over time. One study has found that adults who increased their nighttime sleep from short (<6 hours) to adequate (7-8 hours) over a 6-year study period experienced significantly less visceral fat accumulation over the same time (Chaput, Bouchard, & Tremblay, 2014).

Recently, there has been an initiative to delay school start times to improve sleep duration in adolescence. The American Academy of Paediatrics (AAP) published a policy statement in 2014 recommending middle and high schools in the United Stated delay the start of class to 8:30 am or later. The AAP argues that delaying school start times will help to align school schedules to the biological clocks of adolescents, whose circadian rhythms naturally shift up to two hours later at the start of puberty. Some evidence has shown that delaying school start times by 30 minutes can increase sleep duration by 45 minutes, resulting in significant improvements in measures of mood and alertness (Owens, Belon, & Moss, 2010). However, to my knowledge, no studies have examined the impact this may have on eating behaviour and weight gain in this group. The implementation of later school start times may offer a unique natural experiment to help understand the clinical significance of sleep extension to eating behaviour and weight control in adolescence.

10.5.2 Beyond sleep duration: sleep timing, circadian misalignment and sleep architecture

As outlined in Chapter 1, sleep is a multidimensional construct. While the focus of this thesis was sleep duration, a comprehensive understanding of the relationship between sleep and overweight will require future research to examine dimensions of sleep beyond duration. Although they may overlap, different dimensions of sleep (sleep disturbances, sleep timing, or sleep patterns) could have distinct roles in the development of obesity.

Some work has begun to understand how various dimensions of sleep behaviour independently associate with weight in children. For example, one study in children aged 12 years found that poorer sleep quality, more sleep disturbances, and a later sleep phase were associated with greater percent body fat, independent of nighttime sleep duration

(Jarrin, McGrath, & Drake, 2013). Sleep timing in particular may be an important factor to consider in relationship between sleep and adiposity in young children. In a study of 4-5 year old children, those children going to bed after 9 pm were found to have an increased risk of obesity and weight gain over a 1 year follow-up period (Scharf & DeBoer, 2014). Similarly, in a sample of preschool children aged 4 years, sleep timing was found to moderate the association between sleep duration and BMI, such that the association was only present among children going to bed after 9 pm (Miller et al., 2014). Indeed, the findings from Study 3, which suggest that shorter sleepers consume additional calories at night, support the idea that sleep timing may be an important dimension to consider in future analyses.

A related concept to sleep timing is circadian misalignment. This refers to a disequilibrium between the internal and social clock which forces many activities out of sync with the body’s natural circadian rhythm (Scheer, Hilton, Mantzoros, & Shea, 2009). One example of circadian misalignment is feeding at night when the internal biological clock is primed for sleep (Gonnissen et al., 2013). Although circadian misalignment probably reduces total sleep time, existing evidence in adults suggests that it may also disrupt metabolic signalling in a way that favours fat accumulation, independent of total sleep time (Ekmekcioglu & Touitou, 2011; Scheer et al., 2009). For example, circadian misalignment either by phase advance or phase delay has been shown to adversely impact glucose-insulin metabolism in adults in experimental studies (Gonnissen et al., 2013). Meal timing in particular has been strongly implicated in metabolic health. In one randomised crossover trial, later meal timing in adults was associated with decreased resting energy expenditure and thermal effect of food, as well as impaired glucose tolerance and daily cortisol secretions (Bandín et al., 2015). There is also some evidence that later meal timing may lessen the effectiveness of interventions to induce weight loss (Garaulet et al., 2013). Although sleep timing has been related to adiposity risk in children (discussed above, and see: Golley, Maher, Matricciani, & Olds, 2013), future work must begin to understand the impact of circadian misalignment in children more specifically.

During sleep the brain oscillates between different sleep phases in successive cycles (Chapter 1, section 1.3.2). Little work to date has examined the impact of chronic short sleep or sleep loss on sleep architecture. Some initial findings suggest that moderate restriction of nighttime sleep (7 hours in bed) decreases the amount of time spent in REM and stage 2 sleep, while time spent in SWS is preserved, supporting the idea that SWS is essential for restoration (Gonnissen, Mazuy, et al., 2013). Interesting, some recent work has shown that inter-individual differences in sleep architecture during a normal night of sleep are associated with parameters of energy balance. For example, one study of 13 healthy adults demonstrated that individual variation in the percentage of time spent in REM sleep was inversely associated with cortisol levels and insulin sensitivity (Gonnissen, Mazuy, et al., 2013). In another study of 16 healthy men, higher levels of SWS were inversely associated with self-reported hunger, cortisol concentrations and glucose sensitivity after a 48 hour stay in a respiration chamber where participants were fed to maintain energy balance. In the same study, the mean amount of REM sleep was positively correlated with both ghrelin and cortisol levels (Rutters et al., 2012). Importantly, these findings suggest that sleep architecture may play a role in the regulation of body weight. This presents an interesting avenue for future research, although this work requires electroencephalography or polysomnography to monitor sleep so demands specialist equipment that can be both costly and time consuming.

There are of course many dimensions of sleep that may be important to consider in the sleep-weight relationship. A review of existing research is out of the scope of this thesis. However, it should be emphasised that including sleep measures beyond duration will help future work to more precisely capture the relationship between sleep and risk of obesity. Furthermore, clearly defining this relationship will be necessary to understand how to optimise sleep behaviour in order to ameliorate risk of excess weight gain.

10.5.3 Biological parameters

The use of Gemini data in this thesis allowed for an investigation of the behavioural pathways through which sleep may influence adiposity. However, it will be important for future work to also investigate the neurophysiological and metabolic pathways mediating this relationship. There is currently very limited work in this area, particularly in children.

10.5.3.1 Neurophysiological pathways to food responsiveness

One interesting avenue for future research will be to understand the neurophysiological pathways by which sleep may enhance food responsiveness in children. In adults, sleep restriction has been found to increase activation in reward and food-sensitive centres of the brain in response to images of unhealthy foods (e.g. crisps). Increased activation has observed in the superior and middle temporal gyri, middle and superior frontal gyri, left inferior parietal lobule, orbitofrontal cortex, and right insula, and this pattern of activity was associated with increased ratings of food desire (Greer et al., 2013). Whether or not similar brain regions are at play in children, and whether this can help to explain the inverse association between sleep and food responsiveness observed in Study 4 (Chapter 8) is not yet known. This is an important avenue for future research given that early childhood reflects a period of rapid development (Giedd et al., 1999), when environmental exposures such as sleep duration may shape future brain development (Beebe, 2011).

10.5.3.2 Neuroendocrine pathways to food responsiveness

Future work must also continue to examine the neuroendocrine pathways through which shorter sleep may favour food consumption. Although existing studies have generated mixed findings (reviewed in Chapter 2, section 2.3), metabolic signals are acutely sensitive to changes in energy balance; so contrasting findings could be due to differences in study protocols, for example, when blood samples were taken, or if and when *ad libitum* feeding was permitted.

Although homeostatic feeding is often discussed as being under the control of metabolic signals such as ghrelin, ghrelin has also been found to modulate brain activity in areas involved in food reward. For example, one study in 12 healthy adults found that intravenously administering ghrelin during an fMRI task, significantly increased activity in reward centres of the brain (amygdala, orbitofrontal cortex, anterior insula, and striatum) in response to images of palatable foods. Furthermore, these responses correlated with self-reported ratings of hunger, suggesting that ghrelin may be implicated in hedonic pathways to food consumption (Malik, McGlone, Bedrossian, & Dagher, 2008).

Interestingly, the most recent sleep restriction study found that levels of ghrelin, but not leptin, increased after a period of insufficient sleep (4 nights of 4.5 hours in bed) (Broussard et al., 2015). During the sleep manipulation, healthy participants were given a standardised diet to maintain energy balance. However, an *ad libitum* snack bar, and lunch and dinner buffets were provided following each sleep phase. Relative to the rested condition (4 nights of 8.5 hours in bed) participants consumed 320 kcal more from carbohydrate-based snacks after sleep restriction, without differences in energy intake from meals. Importantly, the increase in evening ghrelin during sleep restriction was significantly associated with energy intake from sweets during the subsequent phase of *ad libitum* feeding (Broussard et al., 2015). These findings suggest that ghrelin may play a role in hedonic consumption associated with shorter nighttime sleep. This provides an important avenue for future research; in particular, further work should examine whether similar effects are observed with chronic insufficient sleep, particularly in early life.

10.5.3.3 Stress response

Sleep deprivation is itself a physiological stressor (Meerlo, Sgoifo, & Suchecki, 2008). Although the relationship between sleep and the stress systems is likely to be complex and bidirectional (Buckley & Schatzberg, 2005), understanding the role of stress in the sleep-weight relationship reflects an important area for future research, particularly in children.

Stress exposure is known to activate the hypothalamic pituitary axis (HPA) which in turn induces the release of cortisol, a steroid hormone (Müssig, Remer, & Maser-Gluth, 2010), and acute total sleep deprivation in adults has been shown to increase the secretion of cortisol (Wright et al., 2015). Importantly, cortisol is known to enhance visceral fat accumulation (Müssig et al., 2010), so it is possible that sleep-related HPA axis activation plays a role in the abdominal adiposity associated with shorter sleep (Bouchard, 2007; Chaput et al., 2014). However, research is required to clearly define how stress exposure and cortisol release impacts fat accumulation and eating behaviour in shorter sleeping children. This is particularly pertinent in light of evidence presented in this thesis demonstrating an association between sleep and home chaos (Study 5, Chapter 9). Chaos in the home has been used as a proxy for stress exposure in early life, and one recent study demonstrated that it was associated with increased cortisol secretion and higher food responsiveness in pre-school aged children, although sleep was not investigated (Lumeng et al., 2014). The stress-response may therefore play a crucial role in the relationship between sleep, food responsiveness and adiposity in early life, reflecting an exciting avenue for future research.

10.5.4 Sleep and genetic risk of obesity

Body mass is highly heritable and multiple loci have been associated with BMI in genome-wide association studies (Speliotes et al., 2010). However, adiposity results from a complex interaction between genetic, environmental, and behavioural factors (Bouchard, 2008). If short sleep duration is causally implicated in the development of adiposity, then it may be important to consider whether people with shorter sleep durations are more susceptible to genetic effects on adiposity. Viewed differently, individuals with a greater genetic predisposition to obesity might be more susceptible to the deleterious effects of short sleep.

Existing work in this area is limited. One study has used adult twins to examine whether sleep duration alters the proportion of the variance in body weight that can be attributed to genetic factors (the heritability of BMI)(Watson et al., 2012). This study, which included 1,088 twin pairs, found that the heritability of BMI was much higher at shorter sleep durations. For twin pairs averaging <7 hours a night, the heritability of BMI was 70%, whereas for those averaging >9 hours a night heritability was 32%. While it is not possible to tell which pathways are involved from this data, it may be that short sleep provides a permissive environment for the activation of genes that promote obesity (Watson et al., 2012).

A similar question has been examined using molecular genetic data to test whether specific alleles for the obesity associated genes FTO, TMEM18 and NRXN3 alter the susceptibility of children to gain weight with short sleep (Prats-Puig et al., 2013). In this study of 297 children aged 5-9 years, common variants in these genes were found to alter children's vulnerability to obesity measured via BMI, waist circumference and visceral fat. Shorter sleep was related to obesity associated traits only among children homozygous for the nonrisk alleles of FTO (TT genotype), or among children with risk alleles for TMEM18 and NRXN3 (Prats-Puig et al., 2013). Given that genetic risk for obesity is known to operate in part through eating behaviour (Carnell & Wardle, 2007), understanding the role that genotypes may have in the relationship between sleep, hedonic eating and weight could also provide an interesting avenue for future research.

10.6 Concluding thoughts

The work presented in this thesis has helped to better understand sleep in early life, and the behavioural pathways through which it may influence the development of adiposity. The findings highlight the importance of an early bedtime, and suggest that in early childhood shorter sleep may lead to a greater propensity to over-consume. Shorter sleeping toddlers may consume more because of parents' inclination to feed to soothe at night, but changes in food responsiveness may increase food intake and weight in older children with a greater autonomy over their eating behaviour. Living in a more permissive home food environment may also strengthen the impact of sleep on weight, supporting the notion that excess food consumption drives weight gain at this age. Although there are significant limitations, the findings presented in this thesis have clear implications, and pave the way for new research to further understand the behavioural and physiological pathways through which sleep increases the risk of adiposity in early life.

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Appendix A Literature review: Sleep and eating behaviour

1.1 Search Strategy

- "Sleep AND (infant OR child OR adolescent) AND (eat* OR energy OR calori*)"
- Search was limited to English language articles published in peer review journals
- Search generated 592 articles – the title and abstract of which were scanned
- 66 full text accessed to assess eligibility
- 18 articles met criteria and were coded (form below) – these were considered too diverse to synthesise

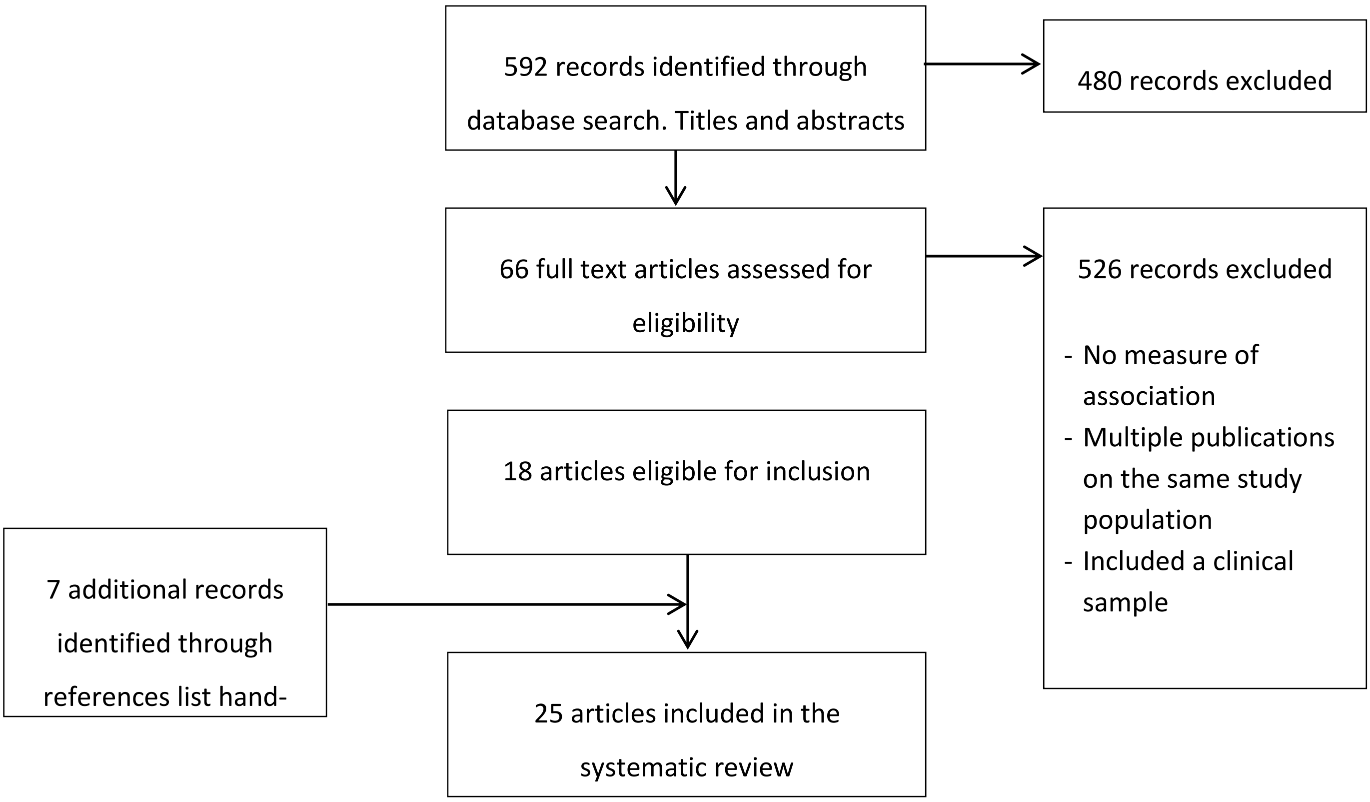


Figure A.1. Flowchart overview of systematic search

2.1 Literature review summary table

	Author, Journal	Design	Sample + Nationality	Objective	Measure	Findings
1	Diethelm (2011) *Clinical Nutrition*	CS	595 1.5y, 2y Ger many	Is nutritional composition of evening meal ass. with sleep duration	• Diet: 3-days weighted diet diary • Sleep: parent reported sleep duration + sleep problems Defined as short (9.5-12), moderate (12.5-13.5) and long (14-16.5) • Night eating = calories consumed between 9pm and 6am	• Higher EI of evening meal ass. with longer sleep duration • Carbs, especially from high GI foods asspcoated with longer sleep • Higher % of short sleepers reported night eating (p=.002) • Absolute EI of the evening meal was lowest in the shortest sleeping group • Daily EI did not differ across SD groups • When ss had nightly eating, the effect of CHO on SD was diminished
2	Bel (2013) *Br J Nut*	CS	1522 12.5-17.5yrs	Examine r'ship between sleep and diet quality using an index of diet quality and considering sex differences	• Sleep: self-report • Diet: 2x 24h recall • Diet quality: diet quality index	• SD and dietary quality score positively correlated • Conclusion: health consequence of insufficient sleep may be mediated by poor dietary quality
3	Hitze (2009) *EJCN*	CS	414 6.1-19.9 yrs	Impact of SD on nutrition status in children/adolescents (and other things)	• Sleep: self-report • Diet: FFQ, nutrition quality score calculated	• Longer sleep associated with healthier diet in girls – short sleeping girls consumed more fast food (and sweets?) • Sort sleeping boys consumed more soft drinks
4	Chaput (2011) *Can J Pub H*	CS	550 Mean = 9.6y	Determine whether EI or PA mediate association between sleep and adiposity	• Sleep: 7 days accelerometry – 4 SD groups • Diet: 3x 24h food recall	• EI, snacking, SSB, fat intake, FV intaje did not differ between SD groups • Adding total EI to sleep-ad model did not change association substantially

	Author, Journal	Design	Sample + Nationality	Objective	Measure	Findings
5	Garaulet (2011) *IJO*	CS	3311 12.5-17.5y	To investigate if inadequate food habits underlie sleep-adiposity association (among others)	• Food habits: FFQ • Sleep: self reported • PA: acclerometer	• Proportion who ate adequate amounts of fruit, veg, fish was lower in shorter sleepers • Probability of have adequate food habits lower in shorter sleepers • Short sleepers more sedentary •
6	Al-Disi (2010) Endocrine Journal	CS	126 F (62 lean, 62 obese) 14-16 y		• Sleep: self-reported • Diet: self reported	• Long sleep associated with better diet • <5h sleep, associated with higher carb intake • Energy intake decreased with decreasing sleep duration, but did not reach statistical sig
7	Chalal (2012) Pediatric Obesity	CS	3398	to assess whether night access to electronics is associated with sleep, diet etc	• Sleep: slelf report • Diet: FFQ	• Diet quality was not associated with sleep duration in lin reg
8	Chen (2006) BMC public health	CS	656 13-18y`	Examine relationship between adequate sleep and health behs	• All self report • Diet – adolescent health promotion scale (eg eating breakfast, 3 meals a day. Drink eater etc)	• Significant negative relationship between low sleep and adopting a healthy diet
9	Clifford (2012) Sleep medicine	CS	41 obese preschool children	Examine association between SD, weight, CI in obese preschool children	• Sleep: diary • Diet: 3x 24h dietary recall	• Longer sleep associated with lower CI postreatment (independent of pretreatment CI) • Each hour of sleep associated with 186 less kcal a day
10	Golley (2013)	CS	2200 9 -16yrs	To examine whether sleep timing is associated with EI	• Sleep: self reported. 4 categories – EE, EL, LE, LL	• EI was linearly associated with sleep duration • LL had lower diet quality than EE

	Author, Journal	Design	Sample + Nationality	Objective	Measure	Findings
	IJO			and diet quality	• Diet: 2 x 24 food recall – calculated diet quality score	• Late bedtime associated with higher intake of energy-sense, nutrient poot foods • Early bedtime – more FV • Findings were independent of sleep duration
11	Kjeldsen (2013) IJO	CS	676 8-11yrs	To examine whether obj measured SD and parent reported sleep problems ass with dietary risk factors	• Sleep: actigraphy, CSHQ • Diet: 7 day web based food record	• SD neg associated with ED of diet (.003), added sugar, SSB • CSHQ score associated with ED • Sleep variability ass with SSB • Independent of confounders (PA, ehtni, screen time, edu, sex, age)
12	Beebe (2013) Sleep	Exp	41 14-16yr	To examine sleep and dietary intake in adolescents using an experimental sleep restriction protocol	• Sleep: actigraphy • Deit: 24h diet recall interviews following exp condition	• After sleep restriction – diet higher in GI and GL & trend towards more kcals and carbohydrates • No difference in protein intake • Sleep restriction also associated with greater consumption of desserts and sweets
13	Adamo (2013) Sleep disorders and therapy	CS	26 obese 13.6 mean	To examine if sleep timing is associated with EI, PA, Sed beh	• Sleep: self-report, categorised as late and normal sleepers • Diet: 3 day diary	• Sleep duration was not significantly difference between late and normal sleepers • EI was 27% higher in short sleepers (425kcals) • (whole sample) late sleep timing associated with higher EI, independent of BMI, age, sex, SD • Small sample, so not significant, but showed tendency toward greater food intake in the evening (late sleepers)
14	Weiss (2010) Sleep	CS	241 17.7 mean (16-19y)	To examine relationship between sleep duration and energy consumption (also	• Sleep: wrist actigraphy, daily sleep log 5-7 nights (short <8h) • Diet: 2 x 24h food recall	• Sleep <8h higher proportion of kcal from fat, lower from carb • <8h sleep associated with 2.1 increased odds of

	Author, Journal	Design	Sample + Nationality	Objective	Measure	Findings
				looked at timing of nutrient intake)	questionnaire – energy consumption was assessed at 3 time periods during the day	consuming 475kcals from snacks • Short sleepers consumed more food in the early morning (5-7am) • Higher total EI in short sleepers (245 difference)
15	Moriera (2010)	CS	1976 5-10yrs	Describe association between food patterns and sleep (among other things)	• Sleep: subjective, hours per day and categorised into 3 • Diet: FFQ	• Longer sleep associated with dietary patterns that included fruits and veg • Longer sleep duration associated with dietary patterns characterised by foods of plant oragine • Shorter sleep duration associated with dietary patterns characterised by foods rich in fat and added sugar (adjusted for age and ei)
16	Westerland (2009) Br J of nutrition	CS	1265 9-11y	Examine whether there is an association between sleep and food consumption patterns	• Sleep: self-report, school and weekend sleep, tiredness • FFQ – 16 items, groups – used factor analysis to split into 2 dietary patterns (energy dense, nutrient rich)	• Boys with shorter sleep and who felt tired during the day were more likely to consume energy-dense food • Girls with shorter sleep consumed more energy dense and less nutrient rich food • Adjusted for PA and screen time weakened associations • Associations stronger for boys
17	Landis (2008) Journal of nursing scholarship	CS	85 15.6 mean 76% african American	Examine associations between TST, hunger, satiety, food cravings, caloric intake	• Sleep: 7 day sleep diary • Diet: 7 day hunger and satiety diary, 24h food recall interview	• Greater food craving score associated with increased daytime sleep
18	Klingenber	Exp	21 males	Investigate effects of 3	• Sleep: controlled as per	• Short sleepers consumed 13% less energy in the

	Author, Journal	Design	Sample + Nationality	Objective	Measure	Findings
	g (2012)	2 conditions each for 3 nights (SS vs LS)	15-19yrs (16.8 mean)	nights of sleep restriction on components of energy balance	condition (4h vs 8h), polysomnography, PSQI • Diet: ad libitum EI (lunch test meal 13:00 day 4) – given standardised breakfast before ad libitum meal, appetite sensations • PA: 24h EE, spontaneous PA • Eating behaviour: 2 factor eating questionnaire	aslibitum meal and expressed a decreased motivation to eat • Large individual variablitly in EI (7 of 21 participants increased EI in SS) • Measured up to 18.00 on day 4 (SS may have compensated at night follow SR) • Also we think SS consume more at night during SS, but in exp condition EI was controlled until 4
19	Tatone (2012) J sleep research	CS – but L association s with weight	1106 6y	Sex differences in the effect of slsep duration and dietary intake were examined prospectively in relation of ow/ob	• Sleep: parent report, open ended q – sleep categorised • Diet: FFQ, eating behaviours, developed 7 food groups	• Shorter sleep associated with less favourable dietary intake at age 6 • Boys: ss consumed FV less, meats more frequently • Girls: ss FV milk less, soft drinks more • Boys: more likely to eat at irregular hours and to eat too much, too fast • These eating behaviours (and not dietary intake mediated the associated between ss and ov/ob in boys) • SD did not ass with problem eating in girls • Concl → shorter sleep ass with problem eating behaviours in boys and diet quality in boys and girls •
21	Awad (2013)	Cross sectional	319 cauc & hispanic	Evaluate the relationship between sleep architecture,	• Sleep: Questionnaire and home polysomnography	• Total sleep time not associated with any dietary variables (kcals, protein, animal fat, veg fat, total

	Author, Journal	Design	Sample + Nationality	Objective	Measure	Findings
	Sleep Breathing **		10-17yrs Tu-CASA study - USA	diet and exercise	• Diet: FFQ, self administered	fat, carbs) but the sample was very small
22	Collison (2010) BMC Pub Health	Cross sectional	9433 10-19 years Saudi Arabia	Examine dietary habits in relation to BMI together with exercise and sleep patterns	• Sleep: question – normal sleep duration • Diet: FFQ • Simple correlations of diet and sleep variables	• Sleep duration negatively correlated with SSCB consumption and positively correlated with fruit consumption
23	Yuasa (2008) J med Inves	Cross-sectional	3291 Japan 1st and 4th grade elementary, 1st grade junior high (no age given)	Evaluated factors associated with the prevention of obesity and the development of healthy habits	• All self reports (parents filled in 30 item questionnaire for children in elementary school)	• Longer sleep duration in children who ate with their family every day (versus not every day)
24	Drescher (2011) J clin sleep med **	Cross sectional	319 caucasion & Hispanic 10-17years	To investigate associations between sleep duration and obesity incidence and risk factors among pre-adolescents and adolescents	• Sleep: parent reported – sleep habits questionnaire • Diet: self-administered FFQ	• Parent reported total sleep time was not associated with any of the examined nutritional variables

	Author, Journal	Design	Sample + Nationality	Objective	Measure	Findings
			Tu-CASA cohort, USA			
25	Hart (2013) Pediatrics	Exp.	37, 8-11year olds USA	To examine the effects of experimental changes in children's SD on self-reported food intake, appetite hormones, weight	• Diet: 3x 24h food recalls per week • (also measured food reinforcement and fasting leptin and grehlin) • Sleep: experimentally manipulated – measured by actigraphy, sleep diary (sleep extension, loss achieved by moving bedtime)	• Difference of 2h 21m between the increase and decrease sleep conditions • During increase – children consumed 134kcal/d less → most of the additional caloric intake (103kcal) came from the additional 3 h that children were awake - also exhibited lower fasting leptin levels (weight was .22kg lower in the sleep extension phase) • No differences in food reinforcement or fasting ghrelin
					•	•

3.1 Coding table

A.) CHILD STUDIES

Dietary Behaviour	Positive association (+)	Inverse association (-)	No association (0)
Energy Intake		(Clifford et al., 2012)	(J.-P. Chaput et al., 2011; Diethelm, Remer, Jilani, Kunz, & Buyken, 2011)
Energy intake, evening meal	(Diethelm et al., 2011),		

Dietary Behaviour	Positive association (+)	Inverse association (-)	No association (0)
Diet quality score	(Chahal et al., 2013)		
Fat intake	(J.-P. Chaput et al., 2011)		
Carbohydrate intake			(J.-P. Chaput et al., 2011)
Protein intake			
Fruit and Veg		(Tatone-Tokuda et al., 2012)(B/G), (Moreira et al., 2010)	(J.-P. Chaput et al., 2011)
Meat consumption		(Tatone-Tokuda et al., 2012)(B)	(Tatone-Tokuda et al., 2012)(G)
Fish			
Milk			
Milk products	(Tatone-Tokuda et al., 2012)(G),(Moreira et al., 2010)		(Tatone-Tokuda et al., 2012)(B)
Cheese			
Soft drinks/ sugar sweetened beverages		(Tatone-Tokuda et al., 2012) (G), (Kjeldsen et al., 2013)	(Tatone-Tokuda et al., 2012)(B) (J.-P. Chaput et al., 2011)
Added sugar		(Kjeldsen et al., 2013)	
Bread/Grains			(Tatone-Tokuda et al., 2012)(B/G)
Breakfast cereals			
French fries			
Crisps			
Sweets			

Eating behaviours	Positive association (+)	Inverse association (-)	No association (0)
Eating at irregular hours		(Tatone-Tokuda et al., 2012)(B)	
Nighttime eating		(Diethelm et al., 2011)	
Eating too much/fast		(Tatone-Tokuda et al., 2012)(B)	
Snack consumption			(J.-P. Chaput et al., 2011)
Meal consumption			

Dietary Behaviour	Positive association (+)	Inverse association (-)	No association (0)
Energy-dense foods/energy density of diet		(Westerlund et al., 2009)(B/G), (Moreira et al., 2010), (Kjeldsen et al., 2013)	
Nutrient rich	(Westerlund et al., 2009)(G)		
Eat with familiy	(Yuasa et al., 2008)(B/G)		
External eating		(Burt et al., 2013)	
Emotional eating			(Burt et al., 2013)
Restrained eating			(Burt et al., 2013)

B.) ADOLESCENT STUDIES

Dietary Behaviour	Positive association (+)	Inverse association (-)	No association (0)

Dietary Behaviour	Positive association (+)	Inverse association (-)	No association (0)
Energy Intake		(Golley et al., 2013), (Weiss et al., 2010)	(Al-disi et al., 2010)
Energy Intake, evening meal			
Diet quality score	(Bel et al., 2013), (M.-Y. Chen, Wang, & Jeng, 2006)		(Golley et al., 2013), (Hitze et al., 2009)
Fat intake	(Al-disi et al., 2010)	(Weiss et al., 2010)	
Carbohydrate intake	(Weiss et al., 2010),	(Al-disi et al., 2010)	
Protein intake			(Weiss et al., 2010), (Al-disi et al., 2010)
Fruit and Veg	(Garaulet et al., 2011), (Collison et al., 2010)		
Meat consumption			
Fish	(Garaulet et al., 2011)		
Milk	(Garaulet et al., 2011)(skim)		(Collison et al., 2010)
Milk products			
Cheese			(Garaulet et al., 2011)
Soft drinks/ sugar sweetened beverages			(Garaulet et al., 2011), (Collison et al., 2010)
Bread/Grains			(Garaulet et al., 2011)
Breakfast cereals	(Garaulet et al., 2011)		(Collison et al., 2010)
French fries			(Garaulet et al., 2011)
Crisps		(Garaulet et al., 2011)	
Sweets			(Garaulet et al., 2011)
Fast food (eg. Pizza, hamburgers, pasta dishes)		(Garaulet et al., 2011)	

Reference list for coding table

1. Clifford et al. (2012) *Sleep Med*.
2. Chaput et al. (2011) *Can. J. Public Health*.
3. Diethelm et al (2011) *Clin. Nutr*.
4. Chahal et al. (2013) *Pediatr. Obes*.
5. Tatone-Tokuda et al (2012) *Sleep Res*.
6. Moreira et al (2010) *Int. J. Env. Res. Pub. Healt*.
7. Kjeldsen et al. (2013) *Int. J. Obes*.
8. Westerlund et al (2009) *Br. J. Nutr*.
9. Yuasa et al. (2008) *J. Med. Investig*.
10. Burt et al. (2013) *Sleep Med*.
11. Golley et al. (2013) *Int. J. Obes*.
12. Weiss et al. (2010) *Sleep*.
13. Al-disi et al. (2010) *Endocr J*.
14. Bel et al. (2013). *Br. J. Nutr*.
15. Chen et al (2006) *BMC Pub Health*.
16. Hitze et al. (2009) *Eur. J. Clin. Nutr*.
17. Garaulet et al. (2011) *Int. J. Obes*.
18. Collison et al (2010) *BMC Pub. Health*.

Appendix B Sleep Questionnaires

4.1 Sleep questions at 15 months

E9.	**When does your child usually go to bed in the evening?**					
	1st born	_____ : _____	(please write hour : minutes e.g. 6:15 pm or 18:15)			
	2nd born	_____ : _____				
E10.	**When does your child usually wake up in the morning?**					
	1st born	_____ : _____	(please write hour : minutes e.g. 6:15 am)			
	2nd born	_____ : _____				
E11.	**How long does you child usually sleep during the daytime?**					
	1st born	_____ hours per day				
	2nd born	_____ hours per day				
E12.	**Do either of your twins usually wake up at night?**	Yes 1st born ☐	Yes 2nd born ☐	Neither ☐		
	If Yes, how often does your child wake up at night?	1st born	_____ times per night		_____ times per week	
		2nd born	_____ times per night		_____ times per week	
	If Yes, for how long does your child wake up at night?	1st born	_____ hours	or	_____ minutes per night	
		2nd born	_____ hours	or	_____ minutes per night	

5.1 Sleep questions at 5 years (including CSHQ)

SLEEP HABITS

The following statements are about your twins' sleep habits. Think about the past week in your child's life when answering the questions. If last week was unusual for a specific reason, choose the most recent typical week. Answer **usually** if something occurs **5 or more times** in a week, **sometimes** if it occurs **2-4 times** in a week, **rarely** if something occurs **never or 1 time** during a week. Also, please indicate whether or not the sleep habit is a problem by circling 'Yes', 'No', or 'Not applicable.

D1. What are your twins' bedtimes: 1st born: ____________________

2nd born: ____________________

D2. Twins' usual amount of sleep each day: (combining night-time sleep and naps) 1st born: ________ hours

2nd born: ________ hours

			Usually (5-7 nights per week)	Sometimes (2-4 nights per week)	Rarely/Never (0-1 nights per week)	Don't Know
D3.	**Child goes to bed at the same time at night**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D4.	**Child falls asleep within 20 minutes after going to bed**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D5.	**Child falls asleep alone in own bed**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D6.	**Child falls asleep in parent's or sibling's bed**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D7.	**Child falls asleep with rocking or rhythmic movements**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D8.	**Child needs special object to fall asleep (doll, special blanket, etc.)**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D9.	**Child needs parent in the room to fall asleep**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐

			Usually (5-7 nights per week)	Sometimes (2-4 nights per week)	Rarely/Never (0-1 nights per week)	Don't Know
D10.	Child is ready to go to bed at bedtime	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D11.	Child resists going to bed at bedtime	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D12.	Child struggles at bedtime (cries, refuses to stay in bed, etc.)	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D13.	Child is afraid of sleeping in the dark	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D14.	Child sleeps with the light on	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D15.	Child is afraid of sleeping alone	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D16.	Child sleeps too little	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D17.	Child sleeps too much	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D18.	Child sleeps the right amount	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D19.	Child sleeps for about the same amount each night	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D20.	Child wets the bed at night	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D21.	Child talks during sleep	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐

			Usually (5-7 nights per week)	Sometimes (2-4 nights per week)	Rarely/Never (0-1 nights per week)	Don't Know
D22.	Child is restless and moves a lot during sleep	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D23.	Child sleepwalks during the night	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D24.	Child moves to someone else's bed during the night (parent, brother, sister, etc.)	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D25.	Child reports body pains during sleep	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
	If so, where?	1st born	________________			
		2nd born	________________			

			Usually (5-7 nights per week)	Sometimes (2-4 nights per week)	Rarely/Never (0-1 nights per week)	Don't Know
D26.	Child grinds teeth during sleep (your dentist may have told you this)	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D27.	Child snores loudly	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D28.	Child seems to stop breathing during sleep	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D29.	Child snorts and/or gasps during sleep	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D30.	Child has trouble sleeping away from home (visiting relatives, vacation)	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐

			Usually (5-7 nights per week)	Sometimes (2-4 nights per week)	Rarely/Never (0-1 nights per week)	Don't Know
D31.	Child complains about problems sleeping	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D32.	Child awakens during night screaming, sweating, and inconsolable	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D33.	Child awakens alarmed by a frightening dream	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D34.	Child awakes once during the night	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D35.	Child awakes more than once during the night	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D36.	Child returns to sleep without help after waking	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D37.	Write the number of minutes in the night your child is awake	1st born	________ minutes		Don't know	☐
		2nd born	________ minutes		Don't know	☐
D38.	Write in the time of day child usually wakes in the morning	1st born	_____ : _____ am		Don't know	☐
		2nd born	_____ : _____ am		Don't know	☐
			Usually (5-7 nights per week)	Sometimes (2-4 nights per week)	Rarely/Never (0-1 nights per week)	Don't Know
D39.	Child wakes up by him/herself	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D40.	Child wakes up with alarm clock	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐

			Usually (5-7 nights per week)	Sometimes (2-4 nights per week)	Rarely/Never (0-1 nights per week)	Don't Know
D41.	**Child wakes up in negative mood**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D42.	**Adults or siblings wake up child**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D43.	**Child has difficulty getting out of bed in the morning**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D44.	**Child takes a long time to become alert in the morning**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D45.	**Child wakes up very early in the morning**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D46.	**Child has a good appetite in the morning**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D47.	**Child naps during the day**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D48.	**Child suddenly falls asleep in the middle of active behaviour**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐
D49.	**Child seems tired**	1st born	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐

During the past week, has your child appeared very sleepy or fallen asleep during the following (check all that apply)?

			Not sleepy	Very sleepy	Falls asleep
D50.	**Playing alone**	1st born	☐	☐	☐
		2nd born	☐	☐	☐

		Not sleepy	Very sleepy	Falls asleep
D51. Watching TV	1st born	☐	☐	☐
	2nd born	☐	☐	☐
D52. Riding in car	1st born	☐	☐	☐
	2nd born	☐	☐	☐
D53. Eating meals	1st born	☐	☐	☐
	2nd born	☐	☐	☐

D54. We hear about 'morning' and 'evening' types of people. Which of these types do you consider your twins to be?

	Definitely a Morning Type	Rather more a Morning than an Evening Type	Rather more an Evening than a Morning Type	Definitely an Evening Type
1st born	☐	☐	☐	☐
2nd born	☐	☐	☐	☐

Appendix C Diet Diary

6.1 Diet diary instruction booklet

PLEASE READ THROUGH THESE INSTRUCTIONS BEFORE STARTING THE FOOD DIARY

We would like you to record in this diary **everything your twins are offered to eat and drink** for **3 days**. Please choose **2 weekdays and one weekend day** and include all food offered **at home and outside the home**.

When to fill in the diary
Please record your twins' diet as you go, **not from memory** at the end of the day. Use written notes on a pad if you forget to take the diary with you. Each diary day covers a 24hr period, so please include any food or drinks that your twins may have been offered during the night. Remember to include foods and drinks between meals (snacks) including water.

It only takes a few minutes for each eating occasion!

Time spent in the care of others
If your twins spend time in the care of others during the recording period then we would very much appreciate if those carers (e.g. other relatives, crèche staff, childminder, friend) would provide details of the food and drink offered.

Please provide the following information for each day of recording:

Day, Date and Time Slots
Please write down the day and date at the top each time you start a new day of recording. Please note the time of each eating occasion into the space provided. For easy use, each day is divided into 3 sections (one on each page), from first thing in the morning to afternoon to evening and through the night.

Record what each twin ate separately
We have provided space in the diary for you to record what each of your twins are offered on a separate page. Please record all of the details requested for each twin. If your twins eat in exactly the same way i.e. time, location, people, food, brand, portion size offered/leftover; please write the details for twin 1 and then record the time for twin 2 and **write 'as above' where the details are identical**, see example on pages 7 & 9.

Where and with whom?

We would like to know where your twins are when they have food or drink. If your twins ate at home, please tell us what room or **part of the house** they were in, e.g. kitchen, living room, and tell us whether they **ate at a table or not** and whether they **were watching television**. If they ate outside the home please write that location down. We would also like to know **who your twins shared meals with**, e.g. whether s/he ate alone, with parents, siblings, or friends.

What do your twins eat?

Please describe the food and drink your twins were offered in as much detail as possible. Include all meals and all snacks. Be specific. The **food and portion guide** will help with the sort of detail we need, like **cooking methods** and any **additions**.

- ***Take-aways and eating out***

  If your twins have been offered **take-aways** or **dishes not prepared at home** such as at a restaurant or a friend's house, please record as much detail about the ingredients as you can e.g. spaghetti with mince, onion and tomato sauce.
- ***Recipes/Homemade dishes***

  If your twins have been offered any **homemade dishes** e.g. sausage casserole, please record this in the recipe section for each day, include the recipe name; ingredients and amounts; number of servings made in total; the cooking method (see examples on pages 12 and 13); how much of the whole recipe each twin was served in the portion size column of the diary (see example on page 8).

Please note the **brand name** (if known). Most packed foods will list a brand name, e.g. Bird's eye, Hovis, or Supermarket own brands. **Labels** are an important source of information for us. It helps us a great deal if you enclose labels from all **ready meals,** labels from **foods of lesser known brands** and any **supplements** your twins take.

Portion size

Instructions for how to record the **portion size offered to and leftover by** (if applicable) your twins can also be found in the **food and portion guide**. In most cases you should be able to follow our 3 step guide to estimate the amount of food offered:

1. If you have used **household measures** (grams/ounces, millilitres/fluid ounces, table/teaspoons) to estimate the food offered to your twins then please record this e.g. *one level teaspoon (tsp) of sugar, 4 heaped tablespoons (tbsp) of peas, 1 inch slice of French bread*. Be careful when describing amounts in spoons that you are referring to the correct spoon size; use photo 20 in the food and portion guide to check.
2. In the absence of the household measurements you may be able to use **packet weights** to estimate amounts eaten e.g. *quarter of a 420g tin of baked beans, one 125g pot of yoghurt*

3. In the absence of household measurements or packet weights please estimate the portion size offered using the **photos of foods** provided in the **food and portion guide**.
   - Write in the food diary the photo number and the size A, B, C, D E, etc. that best shows how much your twins were offered e.g. *3A for a portion of broccoli*
   - The photos could also be used for foods not shown e.g. *pasta is similar to rice, shepherd's pie is similar to lasagne and peas are similar to mixed vegetables.*
   - Remember the **photos are not life size**; the plate is 23 cm; the bowl is 18cm; the fork is 18 cm; the knife is 21 cm.

Please **avoid using subjective terms** (e.g. chunk, mouthful, handful, piece and a little) and instead use standard household measures as much as possible. To **describe any leftovers** please try to use **household measures** (grams/ounces, millilitres/fluid ounces, table/teaspoons) or describe amounts as **a fraction of what was offered** e.g *half (½), quarter ($^1/_4$), third ($^1/_3$), etc.*

Type of eating occasion
Please record the type of eating occasion (as you would define it) e.g. meal or snack, in the diary for each time your twins were offered food or drink.

Was it a typical day?
After each day of recording we ask whether there were any reasons why your twins consumed more or less than usual.

Supplements and medicines
At the end of each recording day please tell us about any supplements or medicines your twins took, including brand, supplement name, strength and amount taken.

Overleaf you can see one day that has already been filled in. This example shows you how we would like you to record your twins' food and drink, for example a meal from a jar and a homemade dish.

Thank you for your time and effort – we really appreciate it!

7.1 Diet diary example entry page

MORNING and EARLY AFTERNOON
Twin 1 Name …*Sally*……………….

Day 1 *Thursday*		Date *04/09/2008*			
Time	**Where? With whom? TV on? At table?**	**Food/Drink description (including BRAND name) & preparation**	**Portion size**		**Meal or snack?**
			offered	**leftover**	
8 am	*Living Room*	*Follow on Milk SMA Progress (1 scoop of powder = 27g)*	*250ml bottle*		*Snack*
	Mother, no TV				
10am	*Kitchen table*	*Weetabix*	*1 biscuit*		*Meal*
	Mother and	*Whole milk Sainsbury's*	*100ml*	*1 tbsp*	
	Sister, TV on	*White sugar Tate and Lyle*	*2 tsp*		
11.30	*Living Room*	*bread (medium cut) Granary from bakers*	*1 slice*		*Snack*
Am	*TV on, Mother*	*margarine Flora light spread*	*medium*		
			spread		
		pure apple juice Sainsbury's	*200ml carton*	*½ of carton*	
1pm	*Kitchen, No TV*	*Chunky Vegetable Risotto Heinz Mum's Own*	*230g jar*	*$^1/_3$ of jar*	*Meal*
	at table, sister,				
	mother and	*Peeled apple*	*1 apple size*	*¼ apple*	
	granddad		*16C*		
		Strawberry and Raspberry Yoghurt Petit Filous	*60g*		

TOP TIP
If any details are exactly the same for twin 2 as for twin 1 feel free to write "as above" in the appropriate sections rather than writing it out twice.

MORNING and EARLY AFTERNOON
Twin 2 Name … *Gemma*………………

Day 1 ***Thursday***		**Date** ***04/09/2008***			
Time	**Where? With whom? TV on? At table?**	**Food/Drink description (including BRAND name) & preparation**	**Portion size**		**Meal or snack?**
			offered	**leftover**	
8.30	*Bedroom,*	*Biscuit for Babies + Toddlers Cow and Gate*	*1 biscuit*		*Snack*
am	*no TV, Mother*				
10am	*As above*	*Rice Krispies Kellogg's*	*Photo 8B*		*Meal*
		Whole milk Sainsbury's	*110 ml*		
11.30	*Garden, no TV*	*Pure apple and blackcurrant juice Heinz*	*60ml juice*		*Snack*
am	*Granddad*	*diluted with tap water*	*240ml*	*¼ of drink*	
1pm	*As above*	*Vegetables with Noodles and Chicken HIPP*	*250g*		*Meal*
		(12months)			
		Peeled apple, sliced	*1 apple size*	*½ apple*	
			16C		
		Frutapura, Plum and Apple Cow and Gate	*100g pot*		

Don't forget to estimate portion sizes for ALL foods and drinks offered.

LATE AFTERNOON & EVENING
Twin 1

Day 1		**Date**			
Time	**Where? With whom? TV on? At table?**	**Food/Drink description (including BRAND name) & preparation**	**Portion size**		**Meal or snack?**
			offered	**leftover**	
4pm	*Lounge, no TV*	*Very weak black tea PG tips (in plastic trainer cup with lid)*	*¾ cup*		*Snack*
	Granddad	*Semi-skimmed milk Sainsbury's*	*¼ cup*	*½ of drink*	
		Fairy cake (see recipe)	*one cake*	*¾ of cake*	
6.15	*Kitchen, No TV*	*Homemade sausage casserole (see recipe)*	*15C*		*Meal*
pm	*at table, Sister*	*Penne pasta, boiled Sainsbury's*	*12B*		
	Mother, Father	*Water Tap*	*100ml*		
8.30	*Bedroom*	*Follow on Milk SMA Progress (1 scoop of powder = 27g)*	*250ml bottle*	*½ of bottle*	*Snack*
pm	*Father*				

Was the **amount of food** or drink that your twins had today about **what they usually have**, less than usual, or more than usual?

TWIN 1

Yes, usual ☐ No, **less** than usual ☑ No, **more** than usual ☐

Please tell us why they had less than usual

She was feeling ill

Please tell us why they had more than usual

TWIN 2

Yes, usual ☐ No, **less** than usual ☐ No, **more** than usual ☑

Please tell us why they had less than usual

Please tell us why they had more than usual

She was at a birthday party

Did your twins **finish all of the food and drink you offered** them today?

Twin 1	Yes	✓	No	
Twin 2	Yes	✓	No	

If no, please **go back to the diary and make a note of any leftovers**.

Did your twins take any **vitamins, minerals, medicines or other food supplements** today?

Yes ✓ No

If yes, for each of your twins **please describe the supplements or medicines they took below**

Which twin?	Brand	Name (in full) including strength	Number of pills, capsules, teaspoons
1	Abidec	Multivitamin syrup with omega 3	5ml (1 tsp)
2	As above	As above	As above

Please record over the page details of any recipes or (if not already described) ingredients of made up dishes or take-away dishes.

Write in recipes or ingredients of made up dishes or take-away dishes			
NAME OF DISH *Fairy Cakes –*		***SERVES:*** *makes 12 cakes*	
Ingredients	***Amount***	***Ingredients***	***Amount***
Tate & Lyle caster sugar	*175g*	*Silver Spoon icing sugar*	*140g*
Anchor butter, unsalted	*175g*	*Yellow food colouring*	*3 drops*
Eggs	*3*	*water*	*2 tablespoons*
Homepride self-raising flour	*175g*		
Baking powder	*1 tsp*		

Brief description of cooking method

Mix together and bake for 15 min.

Mix icing sugar with water and add colouring. Approx. 1 tsp of icing on each cake

8.1 Diet diary general questions

General questions

1. Are either of your twins regularly cared for outside the home?

Twin 1 Yes ☐ No ☐ **Twin 2** Yes ☐ No ☐

If YES are these out-of-home meals prepared by you?

Twin 1 Yes ☐ No ☐ Sometimes ☐ **Twin 2** Yes ☐ No ☐ Sometimes ☐

.

Please list, for an average week, the typical times and frequency that your twins eat outside of the home?
e.g. *at nursery 0900-1700 5 days a week or at grandma's 0800-1400 2 days a week*

Twin 1 ***Please specify***	**Twin 2** ***Please specify***

If your nursery provides a **copy of the nursery menu** for the recording period we would appreciate if you could send this to us when you return the completed diary. This will help us later with the analysis of your twins' diet.

Fats for spreading and cooking

2. Which type of fat spread do you use most often for your twins in the last 3 days? Please record the full product name and fat content. e.g. *Flora Omega 3 plus, low fat spread, 38% fat, polyunsaturated*

Twin 1 ***Please specify***	**Twin 2** ***Please specify***

3. How thickly do you spread butter/margarine on bread/crackers for your twins?

Twin 1 Thick ☐ Medium ☐ Thin ☐ None ☐

Twin 2 Thick ☐ Medium ☐ Thin ☐ None ☐

4. Which type of cooking fat/oil do your household use most often in the last 3 days? Please record the full product name.
e.g. *Sainsbury's sunflower oil*

Twin 1 ***Please specify***	**Twin 2** ***Please specify***

Bread

5. Which type of bread do your twins eat most often in the last 3 days?

Twin 1		Twin 2	
Do not eat ☐	White ☐	Do not eat ☐	White ☐
Granary ☐	Wholemeal ☐	Granary ☐	Wholemeal ☐
Brown ☐	50/50 bread e.g. *Hovis Best of Both* ☐	Brown ☐	50/50 bread e.g. *Hovis Best of Both* ☐
Other ☐		Other ☐	
If other please specify		***If other please specify***	

6. Was it a large loaf or a small loaf?

Twin 1 Large . ☐ Small . ☐

Twin 2 Large . ☐ Small . ☐

7. If the bread was shop bought, how was it sliced?

Twin 1 Thick . ☐ Medium . ☐ Thin . ☐ Unsliced . ☐

Twin 2 Thick . ☐ Medium . ☐ Thin . ☐ Unsliced . ☐

Meat

8. If your twins ate meat in the last 3 days, do they eat the visible fat?

Twin 1 Ate all . ☐ Ate most . ☐ Ate some . ☐ Ate none / Do not eat ☐

Twin 2 Ate all . ☐ Ate most . ☐ Ate some . ☐ Ate none / Do not eat ☐

9. If your twins ate poultry in the last 3 days, do they eat the skin?

Twin 1 Always ☐ Sometimes ☐ Never ☐ Do not eat ☐

Twin 2 Always ☐ Sometimes ☐ Never ☐ Do not eat ☐

Fruit and vegetables

10. If your twins ate apples/pears in the last 3 days, do they eat the skin?

Twin 1
Always . ☐ Sometimes . ☐ Never . ☐ Do not eat . ☐

Twin 2
Always . ☐ Sometimes . ☐ Never . ☐ Do not eat . ☐

11. If your twins ate potatoes in the last 3 days, do they eat the skin?

Twin 1
Always . ☐ Sometimes . ☐ Never . ☐ Do not eat . ☐

Twin 2
Always . ☐ Sometimes . ☐ Never . ☐ Do not eat . ☐

Salt

12. Do you add salt to your twins' food at the table?

Twin 1
Always . ☐ Sometimes . ☐ Never . ☐

Twin 2
Always . ☐ Sometimes . ☐ Never . ☐ .

13. Do you add salt substitute to your twins' food at the table? *e.g. LoSalt*

Twin 1
Always . ☐ Sometimes . ☐ Never . ☐

Twin 2
Always . ☐ Sometimes . ☐ Never . ☐ .

14. Do you add salt to your twins' food during cooking?

Twin 1
Always . ☐ Sometimes . ☐ Never . ☐

Twin 2
Always . ☐ Sometimes . ☐ Never . ☐ .

15. Do you add salt substitute to your twins' food during cooking? *e.g. LoSalt*

Twin 1
Always . ☐ Sometimes . ☐ Never . ☐

Twin 2
Always . ☐ Sometimes . ☐ Never . ☐ .

Special diet

16. Do either of your twins follow a special diet in the last 3 days? *e.g. vegetarian, milk-free, other*

Twin 1 Yes ☐ No ☐ **Twin 2** Yes ☐ No ☐

Please specify	*Please specify*

Breakfast cereals

17. How much milk do your twins usually have on breakfast cereal?

Twin 1 ☐ ml or ☐ fl oz Do not eat ☐ **Twin 2** ☐ ml or ☐ fl oz Do not eat ☐

18. How do you usually make porridge for your twins?

Twin 1 Water only ☐ Milk only ☐ Milk and water ☐ Do not eat ☐

Twin 2 Water only ☐ Milk only ☐ Milk and water ☐ Do not eat ☐

19. Do you usually flavour the porridge?

Twin 1 With sugar ☐ With honey ☐ With salt ☐ Neither / do not eat ☐

Twin 2 With sugar ☐ With honey ☐ With salt ☐ Neither / do not eat ☐

20. How do you usually make instant oat cereal for your twins?

Twin 1 Water only ☐ Milk only ☐ Milk and water ☐ Do not eat ☐

Twin 2 Water only ☐ Milk only ☐ Milk and water ☐ Do not eat ☐

21. Do you usually flavour the instant oat cereal?

Twin 1 With sugar ☐ With honey ☐ With salt ☐ Neither / do not eat ☐

Twin 2 With sugar ☐ With honey ☐ With salt ☐ Neither / do not eat ☐

What drinks do your twins consume?

1) What type of milk do your twins most often drink? ***Tick only one***

	Twin 1	Twin 2		Twin 1	Twin 2
Infant formula	☐	☐	Cows milk (Whole)	☐	☐
Follow-on formula	☐	☐	Cows milk (Semi-skimmed)	☐	☐
			Cows milk (Skimmed)	☐	☐
			Other (e.g. soya or goats milk)	☐	☐

Please specify how formula is made i.e no. of scoops (rounded or flat) of powder and amount of water added or from ready made carton:

Please specify the brand/type if other

2a) What size of beaker/cup do your twins mostly drink from?
You can estimate the size by filling your twins' cups with water to the usual level and emptying the water into a measuring jug

Twin 1 ☐ ml or ☐ fl oz Do not use ☐ **Twin 2** ☐ ml or ☐ fl oz Do not use ☐

2b) What size bottle do your twins mostly drink from?

Twin 1 ☐ ml or ☐ fl oz Do not use ☐ **Twin 2** ☐ ml or ☐ fl oz Do not use ☐

3) When you twins drink pure (100%) fruit juice is it usually diluted with water?

Twin 1 ☐ Yes ☐ No ☐ Do not drink **Twin 2** ☐ Yes ☐ No ☐ Do not drink

If yes, how much juice to water? e.g. *half juice/half water, 150ml juice/50ml water*

Twin 1	Twin 2
Please specify	***Please specify***

4) What type of squash do your twins normally drink?

Twin 1 ☐ Ordinary ☐ No added sugar / low calorie / diet ☐ Do not drink **Twin 2** ☐ Ordinary ☐ No added sugar / low calorie / diet ☐ Do not drink

How much water is usually used to dilute squash? e.g. *half squash/half water, 150ml squash/50ml water*

Twin 1	Twin 2
Please specify	***Please specify***

5 Do your twins drink tea?

Twin 1 NO ☐ YES ☐ **Twin 2** NO ☐ YES ☐

If yes, how much milk is usually added?

Twin 1 ☐ ml or ☐ fl oz Do not add milk ☐ **Twin 2** ☐ ml or ☐ fl oz Do not add milk ☐

If yes, how many teaspoons (see photo 20E in the portion guide) of sugar do your twins have in tea?

Twin 1 Do not add sugar ☐ No of teaspoons ☐ **Twin 2** Do not add sugar ☐ No of teaspoons ☐

6) Do your twins go to bed with a drink/bottle or routinely have a drink/bottle during night time?

Twin 1 NO ☐ YES ☐ **Twin 2** NO ☐ YES ☐

If yes, what is usually in the bottle/drink and how much of it do they drink? e.g. whole cows milk, usually drinks half

Twin 1	Twin 2
Please specify	***Please specify***

When you record the drinks your twins have over the next 3 days you can refer back to this page rather than repeating all the information each time

– <u>except</u> for those occasions where your twins drank something else <u>or</u> from a different container.

9.1 Diet diary portion size guide

- Write in the food diary the photo number and the size A, B, C, D, E, etc. that best shows **how much your twins ate** e.g. *3A for the smallest portion of broccoli*
- The photos could also be used for foods not shown e.g. *pasta is similar to rice, shepherd's pie is similar to lasagne and peas are similar to mixed vegetables.*

1. Fish

2. Baked beans

3. Broccoli

4. Cake

Remember the photos are not life size; the plate is 23 cm; the bowl is 18 cm; the fork is 18 cm; the knife is 21cm

5. Chips

6. Cheese

7. Cornflakes

8. Ice-cream

9. Mixed vegetables

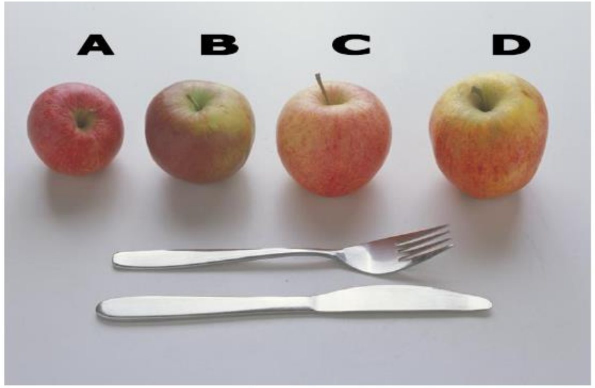
10. Potatoes

11. Pasta

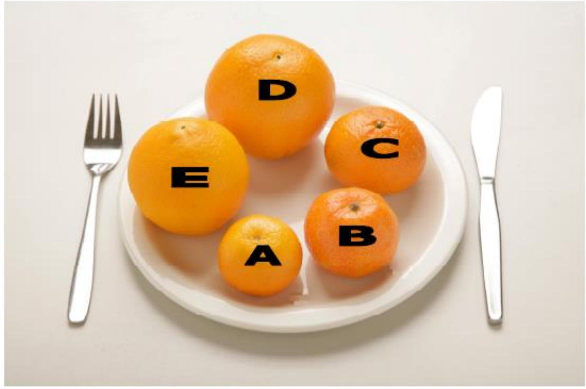
12. Dried fruit

13. Sliced meat

14. Shepherds pie



15. Apples



16. Oranges

17. Bananas

Food	Description & Preparation	Portion size or quantity
Bacon	*Back, middle, streaky; smoked or un-smoked; fat eaten; dry-fried or fried in oil/fat (type used) or grilled rashers*	• Number of slices
Baked beans	*Standard, reduced salt and/or reduced sugar*	• Number of spoons – choose spoon size in photo 20 • Weight on label (e.g. 420g) • Estimate amount with photo 2
Beefburger (hamburger)	*Home-made (ingredients), from a packet (brand name) or take-away; fried (type of oil/fat), microwaved or grilled; economy; with or without bread roll*	• Number of burgers • Weight on pack (e.g. 125g)
Biscuits	*What sort and brand e.g. cheese, wafer, crispbread, sweet, chocolate, shortbread, home-made*	• Number of biscuits • Size (standard or mini variety)
Bread (see also sandwiches)	*Wholemeal, granary, white or brown; currant, fruit, malt; large or small loaf; sliced or unsliced loaf; give brand*	• Number of slices • Thick, medium or thin slices
Bread rolls	*Wholemeal, white or brown; alone or with filling; crusty or soft*	• Number of rolls • Weight on pack per roll (e.g. 50g)
Breakfast cereal (see also porridge)	*What sort and brand e.g. Kellogg's cornflakes; any added fruit and/or nuts; Muesli – added sugar and/or fruit*	• Number of spoons – choose spoon size in photo 20 • Estimate amount with photo 7
Butter, margarine & fat spreads	*Give full product name*	• Thick/average/thin spread • Number of spoons – choose spoon size in photo 20
Cake	*Individual or slice; type and brand; fruit (rich), sponge, fresh cream, butter-cream, iced; type of filling, homemade or bought*	• Number of cakes/slices • Weight on pack (e.g. 30g) • Estimate size with photo 4
Cheese	*Name, brand and type e.g. cheddar, other hard cheese, cream, cottage, soft; low fat*	• Number of slices • Number of spoons – choose spoon size in photo 20 • Estimate amount with photo 6
Chocolate(s)	*What sort e.g. plain, milk, white, fancy, diabetic; type of filling; give brand name*	• Number of bars or pieces • Weight from label (e.g. 24g)

Food	***Description & Preparation***	***Portion size or quantity***
Chips	*Fresh, frozen, oven, microwave, take-away (where from); thick/straight/crinkle/fine cut; type of oil/fat used for cooking give brand name*	• Number of chips • Estimate amount with photo 5
Cream	*Single, whipped, double or clotted; dairy or non-dairy; low-fat; fresh, UHT/Longlife; imitation cream e.g. Elmlea*	• Number of spoons – choose spoon size in photo 20
Crisps	*What sort e.g. potato, corn, wheat, maize, vegetable etc; give brand; flavour; low-fat or low-salt; premium variety e.g. Kettle chips, Walker's Sensations*	• Weight of packet (e.g. 33g) • If not whole packet describe portion consumed (e.g. $^1/_2$)
Custard	*Pouring custard or egg custard; made with powder and milk/sugar, instant, ready to serve (tinned or carton); low fat, sugar free, brand*	• Number of spoons – choose spoon size in photo 20
Egg	*Boiled, fried (type of oil/fat), scrambled (type of fat used, with or without added milk), poached, omelette (with or without filling, type of oil/fat used), etc*	• Number of eggs • Size (large, medium or small)
Fish (including canned)	*What sort and brand e.g. cod, tuna; fried (type of oil/fat), grilled, poached (water or milk) or steamed; with batter or breadcrumbs; canned in oil, brine or tomato sauce*	• Number of spoons – choose spoon size from photo 20 • Weight on label (e.g. 185g) • Estimate amount with photo 1
Fish cakes & fish fingers	*Type of fish; plain or battered or in breadcrumbs; fried, grilled, baked or microwaved; economy*	• Number of fingers/cakes • Weight on pack (e.g. 30g)
Fruit - fresh	*What sort; eaten with or without skin*	• Number or portion of whole fruit e.g. 3 grapes or ½ a plum • Estimate size with photo 12 (dried fruit), 15 (apples), 16 (oranges), 17 (bananas)
Fruit - stewed/canned	*What sort; sweetened or unsweetened; in fruit juice or syrup; juice or syrup eaten*	• Number or portion of whole fruit e.g. 3 grapes or ½ a plum • Number of spoons – choose spoon size in photo 20 • Weight on can (e.g. 227g)
Home-made dishes	please say what the dish is called (record recipe or details of dish if you can in the section provided) and how many persons it serves	• Number of spoons – choose spoon size in photo 20 • Use photos for identical foods (e.g. photo 14 shepherds pie)

Food	***Description & Preparation***	***Portion size or quantity***
Ice cream	*Flavour; dairy or non-dairy; brand name; luxury/premium; added nuts, fruit*	• Number of spoons – choose spoon size in photo 20 • Estimate amount with photo 8
Jam, honey, marmalade	*What sort; type and brand; low-sugar/diabetic; thick-cut; shop bought/homemade*	• Number of spoons – choose spoon size in photo 20 • Spread thin/medium/thick
Meat (see also bacon, burgers & sausages)	*What sort; cut of meat e.g. chop, breast, minced; lean or fatty; fat removed or eaten; skin removed or eaten; how cooked; with or without gravy*	• Estimate amount with photo 18 (sausages); photo 13 (sliced meat) • Number of spoons e.g. mince – choose spoon size in photo 20
Milk	*Brand and type (whole, semi-skimmed, skimmed); fresh, sterilized, UHT, dried; soya milk (sweetened/unsweetened), goats' milk, rice milk; flavoured; fortified with added vitamins and/or minerals; formula milks for toddlers*	• Volume in fl oz / ml *For formula:* • Amount of powder in scoops/spoons - choose spoon size in photo 20 • Amount of water added in fl oz / ml
Nuts	*What sort and brand; dry roasted, ordinary salted, honey roasted; unsalted*	• Weight and/or portion of a single packet e.g ¼ of 100g packet • Number of whole nuts or packets (with weight of packet)
Pie (sweet or savoury)	*What sort and brand; individual or helping; one pastry crust or two; type of pastry*	• Weight of whole pie and/or slice e.g. ¼ of 200g pie
Pizza	*Thin base or deep pan or French bread; topping; brand name and type*	• Weight of whole pizza and/or slice e.g. $^1/_8$ of 190g pizza • Estimate size of pizza with photo 19
Porridge	*Brand name; made with oats or cornmeal or instant oat cereal; made with milk and/or water; with sugar or honey; with milk or cream*	• Number of spoons of dry oats – choose spoon size in photo 20 • Number of packets (including weight) • Volume of milk/water added fl oz/ml
Potatoes (see also chips)	*Old or new; baked, boiled, roast (type of oil/fat); skin eaten; mashed (with butter/spread and with or without milk); fried/chips (type of oil/fat); instant; any additions e.g. butter*	• Estimate amount with photo 5 (chips), 10 (boiled/roast) • Number of spoons e.g. mash – choose spoon size in photo 20
Pudding (see also ice-cream / yoghurt)	*What sort; e.g. steamed sponge; with fruit; mousse; instant desserts; milk puddings*	• Number of spoons – choose spoon size in photo 20 • Weight on packet e.g. 60g • Estimate amount with photo 4 (cake), 9 (Ice-cream)

Food	***Description & Preparation***	***Portion size or quantity***
Ready-made meals	*Please give brand name and full description of product; did it contain any accompaniments e.g. rice, vegetables, sauces; was it chilled or frozen; microwaved, oven cooked, boil-in-the-bag; was it low fat, healthy eating range. Enclose label and ingredients list if possible.*	• Weight on pack • If not whole pack describe portion consumed e.g. $^1/_3$ of 300g lasagne
Rice	*What sort; e.g. basmati, easy cook, long or short grain; white or brown; boiled or fried (type of oil/fat); brand name*	• Number of spoons – choose spoon size from photo 20 • Estimate amount with photo 11 (pasta)
Salad	*Ingredients; if with dressing what sort (oil and vinegar, mayonnaise); brand name of dressing*	• Amount of each component; e.g. number of whole/slices of tomatoes, leaves; cucumber slices, • Number of spoons of dressing – choose spoon size in photo 20
Sandwiches and rolls	*Type of bread/roll (see Bread & Rolls); butter or margarine; type of filling; including salad, mayonnaise, pickle etc. If shop-bought, where from?*	• Number of rolls or slices of bread; • Amount of butter/margarine (on both slices) • Amount of filling
Sauce – cold (including mayonnaise)	*Tomato ketchup, brown sauce, soy sauce, salad cream, mayonnaise; low fat; brand name*	• Number of spoons – choose spoon size from photo 20
Sausages	*What sort; e.g. beef, pork; fried (type of oil/fat) or grilled; low fat; economy; brand name, size; chipolata/cocktail*	• Number of sausages • If not whole sausage describe portion e.g. ½ a sausage • Estimate size with photo 18
Sausage rolls	*Type of pastry e.g flaky, puff, short crust; brand name*	• Number of rolls • If not whole roll describe portion e.g. ¼ a roll • Weight on packet for each roll.
Savoury snacks - in packet	*What sort: e.g. Cheddars, cheese straws, Twiglets, Pretzels; give brand name, standard, mini or multi-pack*	• Weight on pack (e.g. 25g) • If not whole pack describe portion (e.g. ¼ a pack)

Food	*Description & Preparation*	*Portion size or quantity*
Soup	*What sort; give brand name; cream or clear; canned, packet, instant or vending machine, home-made*	• Number of spoons – choose spoon size from photo 20 • Weight on can e.g. 200g
Spaghetti, other pasta	*What sort; fresh/chilled or dried; white, wholemeal; canned in sauce; type of filling if ravioli, cannelloni etc*	• Estimate amount with photo 11 • Number of spoons – choose spoon size in photo 20
Sugar	*Added to cereals, tea, coffee, fruit, etc; what sort; e.g. white, brown, Demerara*	• Number of spoons – choose spoon size in photo 20 • Heaped or level spoon
Sweets	*What sort: e.g. toffees, boiled sweets, diabetic; give brand name*	• Number of whole sweets or packets • If not whole packet describe portion (e.g. ¾ of 33g) • Weight on pack e.g. 5g
Take-away food or food eaten out	*Name of dish and main ingredients if you can. For example, chicken breast or wings, lamb or other type of kebab, type of vegetable in dish, type of sauce, proportion of meat to vegetable etc. Name of a chain restaurant e.g. McDonalds*	• Portion size as sold e.g. small/medium/large • Use photos for identical foods e.g. photo 5 (chips), photo 19 (pizza).
Toddler foods	*Food in jars: brand name and type of food (e.g. vegetable risotto, fruit puree); Dry Foods: brand name and type of food (e.g. dry powder, cereal)*	• Weight on label • If not whole jar describe portion (e.g. ¾ of 160g) *For powdered foods:* • Number of spoons – choose spoon size in photo 20 • Volume of water/milk added fl oz/ml
Vegetables (not including potatoes)	*What sort; how cooked or raw; additions e.g. butter, other fat or sauce*	• Weight on label e.g. 30g • Estimate amount with photo 3 (broccoli) or 9 (mixed vegetables) • Number of spoons – choose spoon size in photo 20
Yoghurt, fromage frais	*What sort: e.g. natural/plain or flavoured; creamy, Greek, low-fat, very low fat/diet, soya; with fruit pieces or just fruit flavoured; twinpot with separate cereal/crumble; fortified with added vitamins and/or minerals; brand name*	• Weight of pot on label e.g. 60g • Number of spoons – choose spoon size in photo 20

Drinks	***Description & Preparation***	***Portion size or quantity***
Tea	*With/without milk (see section on milk); with/without sugar decaffeinated, herb*	• Volume in fl oz / ml
Milk	*Brand and type (whole, semi-skimmed, skimmed); fresh, sterilized, UHT, dried; soya milk (sweetened/unsweetened), goats' milk, rice milk; flavoured; fortified with added vitamins and/or minerals; formula milks for toddlers; ready-made formula or made-up from powder*	• Volume in fl oz / ml *For milk in tea/coffee:* • fl oz /ml or portion of cup e.g. $^{1}/_{2}$, $^{1}/_{3}$, $^{1}/_{4}$ *For formula:* • Amount of powder in g / scoops / spoons - choose spoon size in photo 20 • Amount of water added in fl oz / ml
Milkshake	*What brand; powder/concentrate, dairy or soya, fresh or long life; whole, semi-skimmed, skimmed milk; flavour; fortified with vitamins and/or minerals*	*If shop bought:* • Volume on label e.g. 200 ml • Portion size as sold e.g. small / medium / large *If home-made:* • Number of spoons of powder / concentrate – choose spoon size in photo 20 • Volume of milk/water added in fl oz / ml
Hot chocolate, cocoa malted drinks etc	*Type and brand; standard/low calorie/lite; instant; all water / half milk half water / all milk (see section on milk); any sugar added*	
Squash / cordial / concentrate	*Give brand name & flavour; no added sugar/low calorie/sugar free; "high" juice; added vitamins/minerals*	• Volume in fl oz / ml of concentrate • Volume in fl oz / ml of added water
Carbonated/fizzy	*Give brand & flavour; diet/low-calorie; can/bottle; caffeine free*	• Volume in fl oz / ml • Volume on label (e.g. 250 ml)
Ready to drink	*Give brand & flavour; no added sugar/low calorie/sugar free; % real fruit juice; added vitamins/minerals*	
Fruit – juice (pure)	*Type and brand e.g. apple, orange; freshly squeezed; sweetened/unsweetened; pasteurised or UHT/Longlife; added vitamins/minerals, omega 3*	
Water	*Tap, filtered, bottled: give brand name*	

10.1 Diet diary meal/snack coding frame

MEAL ITEMS	SNACK ITEMS
Pasta, rice, pizza and other cereals Bread - Wholemeal/white/granary - Chapatti - Pitta bread - Tortilla wraps - Crumpets - English muffins - French toast Wholegrain & high-fibre cereals Other breakfast cereals Eggs and egg dishes Meats and meat dishes, excluding processed meat Processed meat - Sausages - Sausage rolls - Pork pies - Burgers - Coated chicken - Scotch eggs Fish and fish dishes, excluding oily fish Oily fish & dishes Cooked vegetables Chips, fried and roast potatoes, fried potato products Other potatoes (boiled, mashed,baked) & grilled potato products Baked beans Soups Non-meat alternatives - Falafel - Quorn Tofu	Biscuits (sweet and savoury), Cakes and pastries - Cheese scones - Cheese pastries - croissants Sweet bread - Hot cross buns - Teacakes - Brioche - Scones - Scotch pancakes - Fruit bread - Milk bread - Malt/fruit loaf - Iced buns Puddings Cream Cheese Yogurts, fromage frais and yogurt drinks Ice cream (dairy and non-dairy) Confectionery Crisps and savoury snacks - Crackers - Breadsticks - Rusks - Rice cakes Nuts and seeds Sugar and preserves Fruit (fresh, canned, cooked, dried), excluding fruit juice Sauces - Condiments - Custard - Dips e.g. hummus Fats (margarine/butter) and oils Soups and sauce Smoothies Raw vegetables - Tomatoes - Carrots - Cucumber Olives

Appendix D Home Environment Interview transcript

GEMINI HOME ENVIRONMENT MEASURE

Section A - GENERAL INFORMATION QUESTIONS

Today's date: __ / __ / 2010

Family ID Number:

(Mark: researcher will type gemini ID: could you then use named contact and twin names, date of birth, street name, house number, post code, town, as highlighted spaces in red)

A1. Please can I speak to <Named Contact>? *(try to speak to main contact but continue anyway)*

If first phone call:
Hello, this is <researcher name> calling on behalf of the Gemini twin study. Instead of a questionnaire, we are carrying out this part of the study over the phone. Is now a good time to talk?

If not convenient, arrange another time that is convenient and record this in the call attempts excel spreadsheet. If the participant doesn't want to do the interview, also record this in the call attempts spreadsheet.

If yes, proceed as below.
We have the twins' names registered as <twin1 name> and <twin2 name>, is that correct and what you would usually call them? Is <twin1 name> the first born twin and <twin2 name> the second born?

If yes to names and birth order: click 'NEXT' button.
If no to names or birth order: check Gemini ID and insert correct names (in the correct order):

twin 1: ……… twin 2 ……………..

I would like to ask you some questions about <twin1 name> and <twin2 name> and your home. Ideally we need to talk to the person who is responsible for the majority of the food shopping and childcare within the home. Do you think you will be in a position to answer these questions?

If FOLLOW-UP phone call:
Hello, this is <researcher name> calling on behalf of the Gemini twin study. We spoke recently and you agreed to take part in a telephone interview. Is now a good time to talk?

1. If OK to talk and speaking to <named contact>: click 'NEXT button'
2. If OK to talk and NOT speaking to <named contact> fill in name below and click 'NEXT' button.

Could I take your name?

First Name ……………….. Last Name ………………...

3. If NOT OK to talk, arrange a convenient time to call back, make a note of this time and click 'BACK'.

Thank you for taking the time to talk, the interview should take around 30 minutes to complete. Just to give you some background, the aim of this interview is to get a picture of the environment young children are growing up in. There are no right or wrong answers so please just answer honestly. If there are any questions you need me to clarify, or any other information you think would be relevant then please feel free to stop me at any time. All your responses will be kept confidential and anonymous.

A2. Please could you confirm the twin's date of birth?
If different, check Gemini ID and insert correct date of birth ____ / ____ / 2007

A3. Please could you confirm your relationship with <twin1 name> and <twin2 name>?

☐ Mother
☐ Father
☐ Guardian
☐ Same sex partner
☐ Grandparent
☐ Nanny
☐ Other, please specify: …………………………………………………

A4. Please could you confirm the following home address? Read out the address below. Change the details if not correct.

Address : <House number> …<Street name> ………………………
<Town>……………………………………………………….

Postcode : <Postcode> ………………………………………………….

Were there any changes? Yes ☐ No ☐

A5. How many adults, including yourself, currently live in your home? Only include people who are aged 18 years or older and who live in your home all of the time.

………….. Adults

A6. Does this include…

Your husband?	Yes ☐	No ☐
Your wife?	Yes ☐	No ☐
Your partner?	Yes ☐	No ☐

For female participants ask: Does this include your husband? If yes, select no for wife and partner. If no, then ask 'your partner?'
For male participants ask: Does this include your wife? If yes, select no for husband and partner. If no, then ask 'your partner?'

If no to all three, skip G4.5 and G4.6.

A7. How many children, under 18 years of age, not including <twin1 name> and <twin2 name>, currently live in your home?

………….. Children

If no other children, skip A8.

A8. Since the birth of <twin1 name> and <twin2 name>, have any additional children joined the household? *If no, skip A9.*

Yes ☐ No ☐

A9. Please can you give the name, date of birth and sex of each additional child? Complete the table below accordingly.

	Child's name	Date of Birth	Sex
1			male ☐ female ☐
2			male ☐ female ☐
3			male ☐ female ☐
4			male ☐ female ☐

Section B - CHILDCARE

The first section is about your twin’s childcare arrangements.

B1. Are your twins usually looked after together?

Yes ☐ No ☐

If looked after together:

B2. What are the regular arrangements for <twin1 name> and <twin2 name> to be looked after, either while you are at work or for any other reasons? For example, do they attend nursery or do you stay at home full time to care for them?

This is an open question. Tick one of the coding options. If the participant says looked after by a relative – clarify whether this is inside or outside the twins’ home.

If the participant mentions more than one arrangement here, make sure you ask about each arrangement in turn.

☐ Stay at home full time to care for the twins.
☐ Partner stays at home full time to care for the twins.

☐ In the twins’ home by grandparent
☐ In the twins’ home by other relative
☐ In the twins’ home by non-relative (including nannies and au pairs)

☐ Outside the twins’ home by grandparent
☐ Outside the twins’ home by other relative
☐ Outside the twins’ home by non-relative (including childminder)

☐ Nursery / Preschool / School
☐ Other, please specify: …………………………………………………….

B3. In general, about how many hours per week do you use this arrangement for <twin1 name> and <twin2 name>?

ENTER WEEKLY HOURS RANGE 0-80. If the participant says it varies, request she/he estimates the average. If the participant says a number of hours plus a half then round down e.g. 15 and 1/2 hours would be 15 hours. Otherwise, round up or down accordingly e.g. 15 and 3/4 hours would be 16. If ‘stay at home full time to care for twins’ is selected, enter 0 hours.

………………………… Hours per week

B4. Do you make any <u>other</u> regular arrangements for looking after <twin1 name> and <twin2 name>?

Yes ☐ No ☐ If No, continue to Section C.

IF Yes

B5. What is the <u>other</u> arrangement for <twin1 name> and <twin2 name>?
Coding options as above: . …………..

B6. In general, about how many hours per week did you use this arrangement for <twin1 name> and <twin2 name>?

ENTER WEEKLY HOURS RANGE 0-80. If the participant says it varies, request she/he estimates the average. If the participant says a number of hours plus a half then round down e.g. 15 and 1/2 hours would be 15 hours. Otherwise, round up or down accordingly e.g. 15 and 3/4 hours would be 16. If 'stay at home full time to care for twins' is selected, enter 0 hours.

………………………… Hours per week

Do you make any <u>other</u> regular arrangements for looking after <twin1 name> and <twin2 name>?

Yes ☐ No ☐ If No, continue to Section C.

Repeat B-5-6 until answer is No.

If looked after separately:

B7. What are the regular arrangements for <twin1 name> to be looked after, either while you are at work or for any other reasons? For example, does <twin1 name> attend nursery or do you stay at home full time to care for <twin1 name>?

This is an open question. Tick one of the coding options. If the participant says looked after by a relative – clarify whether this is inside or outside the twins' home.

If the participant mentions more than one arrangement here, make sure you ask about each arrangement in turn.

☐ Stay at home full time to care for <twin1 name>.
☐ Partner stays at home full time to care for <twin1 name>.

☐ In the twins' home by grandparent
☐ In the twins' home by other relative
☐ In the twins' home by non-relative (including nannies and au pairs)

☐ Outside the twins' home by grandparent
☐ Outside the twins' home by other relative
☐ Outside the twins' home by non-relative (including childminder)

☐ Nursery / Preschool / School
☐ Other, please specify: …………………………………………………….

B8. What are the regular arrangements for <twin2 name> to be looked after, either while you are at work or for any other reasons? For example, does <twin2 name> attend nursery or do you stay at home full time to care for <twin2 name>?

This is an open question. Tick one of the coding options. If the participant says looked after by a relative – clarify whether this is inside or outside the twins' home.

If the participant mentions more than one arrangement here, make sure you ask about each arrangement in turn.

☐ Stay at home full time to care for <twin2 name>.
☐ Partner stays at home full time to care for <twin2 name>.

☐ In the twins' home by grandparent
☐ In the twins' home by other relative
☐ In the twins' home by non-relative (including nannies and au pairs)

☐ Outside the twins' home by grandparent
☐ Outside the twins' home by other relative
☐ Outside the twins' home by non-relative (including childminder)

☐ Nursery / Preschool / School
☐ Other, please specify: ……………………………………………………….

B9. In general, about how many hours per week do you use this arrangement for <twin1 name>?

ENTER WEEKLY HOURS RANGE 0-80. If the participant says it varies, request she/he estimates the average. If the participant says a number of hours plus a half then round down e.g. 15 and 1/2 hours would be 15 hours. Otherwise, round up or down accordingly e.g. 15 and 3/4 hours would be 16. If 'stay at home full time to care for twins' is selected, enter 0 hours.

………………………… Hours per week

In general, about how many hours per week do you use this arrangement for <twin2 name>?

………………………… Hours per week

B10. Do you make any <u>other</u> regular arrangements for looking after <twin1 name>?

Yes ☐ No ☐

Do you make any <u>other</u> regular arrangements for looking after <twin2 name>?

Yes ☐ No ☐ If both no, continue to Section C.

IF Yes

B11. What is the arrangement for <twin1 name>?

Coding options as above: . …………..

What is the arrangement for <twin2 name>?

Coding options as above: . …………..

B12. In general, about how many hours per week did you use this arrangement for <twin1 name>?

………………………… Hours per week

In general, about how many hours per week did you use this arrangement for <twin2 name>?

………………………… Hours per week

Repeat B10-B11-B12 until answer to B10 is No for both twins.

Section C – HOUSE AND NEIGHBOURHOOD

The next few questions are about where you live.

C1. Which of the following options best describes the type of home you live in?
Read out each of the options below.

☐ Flat (which floor……)
☐ Semi-detached house
☐ Terraced house
☐ Detached house
☐ Other, please specify: ……………………………………………

C2. Do you have stairs in your home?
Yes ☐ No ☐

C3. Would you say that your home is on a busy street with lots of traffic?
Yes ☐ No ☐

Any comments on this section (C1 – C3)

I'm now going to ask some questions about how satisfied you are with where you live. For each question please choose a score from 1 to 5. A score of 1 means strongly dissatisfied, 2 means somewhat dissatisfied, 3 means neither satisfied nor dissatisfied, 4 means somewhat satisfied and 5 means strongly satisfied.

To make sure the participant is ranking correctly, for the first question of each set of questions where there are a number of response options, repeat their response back to them e.g. if participant says 5, interviewer says 'so that's 5, strongly satisfied?' etc. If the participant asks what we mean by neighbourhood, say that it is what they perceive their neighbourhood/local area to be.

C4. How satisfied are you with the quality of schools in your neighbourhood? This includes preschool and nursery.

If the participant says that they are strongly satisfied with the preschools in their neighbourhood but strongly dissatisfied with the secondary schools in their neighbourhood, get them to consider this with their response. For example, they may give a neutral score of 3 when taking this into account.

1 2 3 4 5

C5. How satisfied are you with access to entertainment in your neighbourhood such as restaurants and cinemas?
If participants say there aren't any restaurants/cinemas in their neighbourhood, ask them how satisfied they are with this.
1 2 3 4 5

C6. How satisfied are you with the safety of your neighbourhood? By this we mean safety from threat of crime.
1 2 3 4 5

C7. How satisfied are you with the level of traffic in your neighbourhood?
1 2 3 4 5

C8.1 How satisfied are you with the number of food shops in your neighbourhood?
1 2 3 4 5

C8.2 How satisfied are you with the quality of food shops in your neighbourhood?
1 2 3 4 5

C9.1 How satisfied are you with the number of restaurants in your neighbourhood? This includes all types of restaurants, sit-in or take-away. If participants say there aren't any restaurants in their neighbourhood, ask them how satisfied they are with this.
1 2 3 4 5

C9.2 How satisfied are you with the quality of restaurants in your neighbourhood? Again this includes all types of restaurants, sit-in or take-away.
1 2 3 4 5

C10. How satisfied are you with your neighbourhood as a place to raise children?
1 2 3 4 5

C11. How satisfied are you with your neighbourhood as a place to live?
1 2 3 4 5

C12. How easy it is to walk in your neighbourhood, with 1 being not at all easy and 5 being very easy?
1 2 3 4 5

C13. How easy it is to bicycle in your neighbourhood, with 1 being not at all easy and 5 being very easy?
1 2 3 4 5

Section D – PHYSICAL ACTIVITY ENVIRONMENT

The next section is about activity facilities available to you.

D1. Are there any parks or outdoor recreation areas close to your home?
If the participant asks what we mean by ‘close’ say that we mean parks or outdoor recreation areas that they believe are within a reasonable walking distance from their home or a short drive away.
Yes ☐ No ☐ Don’t know ☐ *If no or don’t know skip D2.*

D2. Do you use any of these with <twin1 name> and <twin2 name> on a regular basis? If the participant asks what we mean by regular say that we mean at least every other week.

Yes ☐ No ☐

D3. Are there any in-door recreation centres, for example a gym or indoor soft play close to your home?

If the participant asks what we mean by ‘close’ say that we mean indoor recreation centres that they believe are within a reasonable walking distance from their home or a short drive away.

Yes ☐ No ☐ Don’t know ☐ *If no or don’t know skip D4.*

D4. Do you use any of these with <twin1 name> and <twin2 name> on a regular basis? If the participant asks what we mean by regular say that we mean at least every other week.

Yes ☐ No ☐

D5. Do you take <twin1 name> and <twin2 name> to any other regular play sessions where they can be physically active, for example activity classes or play areas? Activity classes such as ballet, swimming and other places where the twins can be active such as adventure parks, woods etc are included.

Yes ☐ No ☐

D6. Do you have a garden (or outdoor space) that <twin1 name> and <twin2 name> can play in? This includes shared garden space for people living in flats, but does not include park space, even if it is very close to home.

Yes ☐ No ☐

If no skip D7, D8, D10 and D12.

D7. Would you say that your garden (or outdoor space) is small, medium or large? This is a subjective question. The participant should say what they feel the size of their garden is.

small ☐ medium ☐ large ☐

D8. Do you have any usable play equipment such as swings, slides, climbing frames, trampolines in your garden (or outdoor space)? This includes sandpits. Usable means that it is ready to use. For example, swings are well grounded and have chairs.

Yes ☐ No ☐

If yes, what types of play equipment do you have in your garden (or outdoor space)?

D9. Do <twin1 name> and <twin2 name> each have a usable tricycle, bike, scooter or wheeled toy? Usable means that it is ready to use. For example, bikes have tires that are pumped up and chains that are not broken.

Yes (both) ☐ No ☐ Yes <twin1 name> ☐ Yes <twin2 name> ☐

For the next two questions, please choose a score from 1 to 5: 1 means strongly disagree, 2 means somewhat disagree, 3 means neither agree nor disagree, 4 means somewhat agree, 5 means strongly agree.

D10. To what extent would you agree that <twin1 name> and <twin2 name> have adequate room to play actively in your garden or outdoor space? If the participant asks what we mean by 'actively' say anything that involves physically moving about during playing such as running, jumping, or climbing on things.

For the first question, to make sure the participant is ranking correctly, repeat their response back to them e.g. if participant says 5, interviewer says 'so that's 5, strongly agree?' etc.

1 2 3 4 5 (5=strongly agree)

D11. To what extent would you agree that <twin1 name> and <twin2 name> have adequate room to play actively inside the home? Again, if the participant asks what we mean by 'actively' say anything that involves physically moving about during playing such as running, jumping, or climbing on things.

A possible response may be that there is space in some rooms, but not in others. Get the participant to consider this with their response. For example, if there is only space in one room, the answer might be 4, somewhat agree.

1 2 3 4 5 (5=strongly agree)

For the next two questions, again please choose a score from 1 to 5: 1 means never, 2 means rarely, 3 means some of the time, 4 means most of the time, 5 means all of the time.

D12. How often would you say that <twin1 name> and <twin2 name> are allowed to play actively in your garden or outdoor space?

For the first question, to make sure the participant is ranking correctly, repeat their response back to them e.g. if participant says 5, interviewer says 'so that's 5, all of the time?' etc.

A potential response may be that the twins are only allowed to play outside if an adult is present. If play is never restricted within that parameter, tick 5 all of the time. Explanations for D12 and D13 are irrelevant. It might be that participants rarely allow play in the garden because they do not feel that it is safe. This response should remain as 2, rarely.

1 2 3 4 5

D13. How often would you say that <twin1 name> and <twin2 name> are allowed to play actively inside the home?

1 2 3 4 5

Section E – CHILDREN'S ACTIVITY

The next section is about your twin's activity.

E1. Compared to other children of the same age and sex, how physically active are <twin1 name> and <twin2 name>? Please choose a score from 1 to 5 for each child separately: 1 means much less active, 2 means somewhat less active, 3 means about average, 4 means somewhat more active, 5 means much more active.

<twin1 name>: 1 2 3 4 5

<twin2 name>: 1 2 3 4 5

E2. Do you think <twin1 name> gets enough physical activity?

Yes ☐ No ☐

Do you think <twin2 name> gets enough physical activity?

Yes ☐ No ☐

E3. Do you know how many minutes of physical activity per day health professionals recommend for young children?

____ mins (enter 99 if Don't know)

If the participant asks, the answer is 60 minutes per day.

E4. Do you know how many minutes of physical activity per day health professionals recommend for adults?

____ mins (enter 99 if Don't know)

If the participant asks, the answer is 30 minutes per day.

Your child's free time choices

The next questions are about how much <twin1 name> and <twin2 name> enjoy specific activities. I will ask the questions for <twin1 name> first and then repeat the activities for <twin2 name>.

For each activity, please choose a score from 1 to 5: 1 means does not enjoy it at all, 2 means enjoys it a little, 3 means neither likes nor dislikes, 4 means enjoys it a lot, and 5 means loves it. Say not applicable if <twin1 name> or <twin2 name> never does the activity.

How much does <twin1 name> enjoy the following or similar activities? Read each activity choice in turn. Wait for the participant's response before reading out the next activity choice. For the first question, to make sure the participant is ranking correctly, repeat their response back to them e.g. if participant says 5, interviewer says 'so that's 5, loves it?' etc.

E5.1. Doing jigsaws or puzzles:	1	2	3	4	5	NA
E5.2. Drawing and making things:	1	2	3	4	5	NA
E5.3. Watching TV:	1	2	3	4	5	NA
E5.4. Playing computer games:	1	2	3	4	5	NA
E5.5. Riding a bicycle or playing with wheeled toy:	1	2	3	4	5	NA
E5.6. Walking:	1	2	3	4	5	NA
E5.7. Playing ball games such as catch, football, tennis:	1	2	3	4	5	NA
E5.8. Climbing on things:	1	2	3	4	5	NA
E5.9. Running:	1	2	3	4	5	NA
E5.10. Dancing:	1	2	3	4	5	NA

How much does <twin2 name> enjoy the following or similar activities? Again, for each question, please choose a score from 1 to 5 (1 means does not enjoy it at all, 2 means enjoys it a little, 3 means neither likes nor dislikes, 4 means enjoys it a lot, and 5 means loves it) and say not applicable if <twin2 name> never does the activity. Read each activity choice in turn. Wait for the participant's response before reading out the next activity choice.

E6.1. Doing jigsaws or puzzles:	1	2	3	4	5	NA
E6.2. Drawing and making things:	1	2	3	4	5	NA
E6.3. Watching TV:	1	2	3	4	5	NA
E6.4. Playing computer games:	1	2	3	4	5	NA
E6.5. Riding a bicycle or playing with wheeled toy:	1	2	3	4	5	NA
E6.6. Walking:	1	2	3	4	5	NA
E6.7. Playing ball games such as catch, football, tennis:	1	2	3	4	5	NA

E6.8. Climbing on things: 1 2 3 4 5 NA

E6.9. Running: 1 2 3 4 5 NA

E6.10. Dancing: 1 2 3 4 5 NA

Section F – PARENTAL MODELING OF ACTIVITY

The questions in this section refer to the parent(s) or primary caregiver(s) who live in the same home as the twins. This may or may not be the biological parent(s).

For the next section, again please choose a score from 1 to 5. 1 means never, 2 means rarely, 3 means sometimes, 4 means often, 5 means very often. For each question, please indicate whether your response is the same or different for <twin1 name> and <twin2 name>.

Note that scores of <twin1 name> are automatically copied to <twin2 name>. If parent indicates a
difference between twins, score can be adjusted for <twin2 name>. Always score <twin1 name>
first and then <twin2 name>.

F1. How often do you or your <husband/wife/partner> encourage <twin1 name> and <twin2 name> to do physical activity? For the first question, to make sure the participant is ranking correctly, repeat their response back to them e.g. if participant says 5, interviewer says 'so that's 5, very often?' etc. If parents say 1 because they don't need to as their twins are already physically active, still keep response as 1. In other words, it doesn't matter what the reason is.

1 2 3 4 5

If different arrangement for twins: <twin2 name>: 1 2 3 4 5

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

F2. How often do you or your <husband/wife/partner> do physical activity or play sports with <twin1 name> and <twin2 name>?
1 2 3 4 5

If different arrangement for twins: <twin2 name>: 1 2 3 4 5

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.

Is that for <twin1 name> or <twin2 name> or both?

F3. How often do you or your <husband/wife/partner> provide transport to a place where <twin1 name> and <twin2 name> can do physical activity? By this we mean provide transport by car (or other vehicle) rather than by foot.

1 2 3 4 5

If different arrangement for twins: <twin2 name>: 1 2 3 4 5

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

F4. How often do you or your <husband/wife/partner> watch <twin1 name> and <twin2 name> participate in physical activity?

1 2 3 4 5

If different arrangement for twins: <twin2 name>: 1 2 3 4 5

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

F5. How often do you or your <husband/wife/partner> tell <twin1 name> and <twin2 name> that being physically active is good for their health?

1 2 3 4 5

If different arrangement for twins: <twin2 name>: 1 2 3 4 5

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

F6. How often do you or your <husband/wife/partner> try to be active in front of <twin1 name> and <twin2 name>? This includes occasions where the twins see their parent(s) preparing to exercise, even if they are not able to actually see them exercise.

1 2 3 4 5

If different arrangement for twins: <twin2 name>: 1 2 3 4 5

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

F7. How often do you or your <husband/wife/partner> try to show enthusiasm about being active?
1 2 3 4 5

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

F8. How often do you or your <husband/wife/partner> show <twin1 name> and <twin2 name> how much you enjoy being active?
1 2 3 4 5

If different arrangement for twins: <twin2 name>: 1 2 3 4 5

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Section G - MEDIA

The next section is about the media equipment you have in your home

G1. How many working TV's do you have in your home? Include TV's that are temporarily broken if there is a plan to get them fixed.
…….. (enter 99 if Don't know, enter 0 if none) *If 0, skip G2*

G2. Do you have cable or satellite? This does not include freeview.
Yes ☐ No ☐

G3. How many working VCR or DVD players do you have in your home? Include VCR's or DVD players that are temporarily broken if there is a plan to get them fixed. Also include DVD players within computers if they are used to watch films on.
…... (enter 99 if Don't know, enter 0 if none, if 0 to G1 and 0 to G3, skip G4)

G4.1 On average, how long do <twin1 name> and <twin2 name> watch TV or DVDs during the following times of a typical weekday (Monday to Friday) at this time of year?
Only include TV viewing in the home. Write hours and minutes. If less than one hour e.g. 15 minutes put 0 hours and 15 minutes. If 1 hour, put 1 hour and 0 minutes. Note that scores of <twin1 name> are automatically copied to <twin2 name>. If parent indicates a difference between twins, score can be adjusted for <twin2 name>. Always score <twin1 name> first and then <twin2 name>. If participants say that the TV is on for a prolonged period e.g. all morning, but the twins are not always watching it, check whether the twins are in the same room as the TV during this time and record the number of hours that it is on whilst they are in the room. If this happens, make a note in the database changes sheet. For G4.1 – G4.6, read out each of the times (e.g. morning (6am to 12 noon)) in turn and wait for the participant's response before reading out the next time.

Morning (6am to 12 noon) ……. hours ……. minutes per day

Afternoon (12 noon to 6pm) …….. hours ……. minutes per day
Evening (6pm to midnight) ……. hours ……. minutes per day

If different arrangement for twins, enter answers for <twin1 name> above and for <twin2 name> below:

Morning (6am to 12 noon) ……. hours ……. minutes per day
Afternoon (12 noon to 6pm) …….. hours ……. minutes per day
Evening (6pm to midnight) ……. hours ……. minutes per day

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

G4.2 On average, how long do <twin1 name> and <twin2 name> watch TV or DVDs during the following times of a typical weekend day at this time of year?
Only include TV viewing in the home. Write hours and minutes. If less than one hour e.g. 15 minutes put 0 hours and 15 minutes. If 1 hour, put 1 hour and 0 minutes. Note that scores of <twin1 name> are automatically copied to <twin2 name>. If parent indicates a difference between twins, score can be adjusted for <twin2 name>. Always score <twin1 name> first and then <twin2 name>. If participants say that the TV is on for a prolonged period e.g. all morning, but the twins are not always watching it, check whether the twins are in the same room as the TV during this time and record the number of hours that it is on whilst they are in the room. If this happens, make a note in the database changes sheet.

Morning (6am to 12 noon) ……. hours ……. minutes per day
Afternoon (12 noon to 6pm) …….. hours ……. minutes per day
Evening (6pm to midnight) ……. hours ……. minutes per day

If different arrangement for twins, enter answers for <twin1 name> above and for <twin2 name> below:

Morning (6am to 12 noon) ……. hours ……. minutes per day
Afternoon (12 noon to 6pm) …….. hours ……. minutes per day
Evening (6pm to midnight) ……. hours ……. minutes per day

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

G4.3 On average, how long do you watch TV or DVDs during the following times of a typical weekday (Monday to Friday) at this time of year? Only include TV viewing in the home. Write hours and minutes. If less than one hour e.g. 15 minutes put 0 hours and 15 minutes. If 1 hour, put 1 hour and 0 minutes.

Morning (6am to 12 noon) ……. hours ……. minutes per day
Afternoon (12 noon to 6pm) …….. hours ……. minutes per day
Evening (6pm to midnight) ……. hours ……. minutes per day

G4.4 On average, how long do you watch TV or DVDs during the following times of a typical weekend day at this time of year? Only include TV viewing in the home. Write hours and minutes. If less than one hour e.g. 15 minutes put 0 hours and 15 minutes. If 1 hour, put 1 hour and 0 minutes.

Morning (6am to 12 noon) ……. hours ……. minutes per day
Afternoon (12 noon to 6pm) …….. hours ……. minutes per day
Evening (6pm to midnight) ……. hours ……. minutes per day

G4.5 On average, how long does your <husband/wife/partner> watch TV or DVDs during the following times of a typical weekday (Monday to Friday) at this time of year? Only include TV viewing in the home. Write hours and minutes. If less than one hour e.g. 15 minutes put 0 hours and 15 minutes. If 1 hour, put 1 hour and 0 minutes.

Morning (6am to 12 noon) ……. hours ……. minutes per day
Afternoon (12 noon to 6pm) …….. hours ……. minutes per day
Evening (6pm to midnight) ……. hours ……. minutes per day

G4.6 On average, how long does your <husband/wife/partner> watch TV or DVDs during the following times of a typical weekend day at this time of year? Only include TV viewing in the home. Write hours and minutes. If less than one hour e.g. 15 minutes put 0 hours and 15 minutes. If 1 hour, put 1 hour and 0 minutes.

Morning (6am to 12 noon) ……. hours ……. minutes per day
Afternoon (12 noon to 6pm) …….. hours ……. minutes per day
Evening (6pm to midnight) ……. hours ……. minutes per day

G4.7 On average, how long do you watch TV or DVDs as a family on a typical weekday day at this time of year? This includes occasions when it is just <twin1 name> and <twin2 name> and yourself or <twin1 name> and <twin2 name> and your <husband/wife/partner>. A possible response is that they sit down with the twins or are in the same rooms as the twins whilst they are watching TV but they are not watching the TV themselves. This is not included. Parents must also be watching the TV.

Morning (6am to 12 noon) ……. hours ……. minutes per day
Afternoon (12 noon to 6pm) …….. hours ……. minutes per day
Evening (6pm to midnight) ……. hours ……. minutes per day

G4.8 On average, how long do you watch TV or DVDs as a family on a typical weekend day at this time of year? This includes occasions when it is just <twin1 name> and <twin2 name> and yourself or <twin1 name> and <twin2 name> and your <husband/wife/partner>. A possible response is that they sit down with the twins or are in the same rooms as the twins whilst they are watching TV but they are not watching the TV themselves. This is not included. Parents must also be watching the TV.

Morning (6am to 12 noon) ……. hours ……. minutes per day
Afternoon (12 noon to 6pm) …….. hours ……. minutes per day
Evening (6pm to midnight) ……. hours ……. minutes per day

G5. Do <twin1 name> and <twin2 name> share a bedroom?

Yes ☐ No ☐

G6. Do <twin1 name> and <twin2 name> have a working TV in their bedroom? Include TV's if it is a shared bedroom and the TV belongs to another child.

Yes ☐ No ☐

If different arrangement for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name> (and the twins do not share a bedroom), prompt them to check. Is that for <twin1 name> or <twin2 name> or both?

G7. How many working computers or laptops do you have in your home? Include computers or laptops that are temporarily broken if there is a plan to get them fixed. Also include iPads.

…….. (enter 0 if none) *If 0, skip G8*

G8. Do <twin1 name> and <twin2 name> have a computer or laptop in their bedroom? Include computers if it is a shared bedroom and the computer belongs to another child.

Yes ☐ No ☐

If different arrangement for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name> (and the twins do not share a bedroom), prompt them to check. Is that for <twin1 name> or <twin2 name> or both?

G9. How many working games consoles, such as Play Station, Nintendo DS, Wii do you have in your home? Include game consoles that are temporarily broken if there is a plan to get them fixed. This includes hand held games consoles.

…….. (enter 0 if none) *If 0, skip G10, if 0 to G1, G7, and G9, skip G11-G13*

G10. Do <twin1 name> and <twin2 name> have a games console in their bedroom? Include games consoles if it is a shared bedroom and the computer belongs to another child.

Yes ☐ No ☐

If different arrangement for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name> (and the twins do not share a bedroom), prompt them to check. Is that for <twin1 name> or <twin2 name> or both?

G11. Do you have any rules around TV watching or computer use for <twin1 name> and <twin2 name>?

Yes ☐ No ☐

G12. Do you ever reward good behaviour with extra TV or computer time?

Yes ☐ No ☐

If different arrangement for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

G13. Do you ever reduce TV or computer time if <twin1 name> or <twin2 name> is naughty?

Yes ☐ No ☐

If different arrangement for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

G14. Do <twin1 name> and <twin2 name> ever eat while watching TV? This includes meals and snacks that are eaten in front of the TV. If the participant says sometimes, check whether this is on a weekly basis. If not on a weekly basis, enter no.

Yes ☐ No ☐ *If No skip G15-G18*

If different arrangement for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

G15. How many days per week do <twin1 name> and <twin2 name> eat breakfast while watching TV?
Note that for G15-G18 the scores of <twin1 name> are automatically copied to <twin2 name>. If parent indicates a difference between twins, score can be adjusted for <twin2 name>. Always score <twin1 name> first and then <twin2 name>.

0 1 2 3 4 5 6 7

If different arrangement for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

G16. How many days per week do <twin1 name> and <twin2 name> eat a midday meal while watching TV?

0 1 2 3 4 5 6 7

If different arrangement for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

G17. How many days per week do <twin1 name> and <twin2 name> eat an evening meal while watching TV?

0 1 2 3 4 5 6 7

If different arrangement for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

G18. How many days per week do <twin1 name> and <twin2 name> eat snacks while watching TV?

0 1 2 3 4 5 6 7

If different arrangement for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

The next section is about your twin's sleep.

G19. When do <twin1 name> and <twin2 name> usually go to bed in the evening?
Write hour : minutes e.g. 6:15pm or 18:15pm.
Note that for G19-G21 the scores of Mia are automatically copied to Lee. If parent indicates a difference between twins, score can be adjusted for Lee. Always score Mia first and then Lee.
<twin1 name>: …….. : ……..
<twin2 name>: …….. : ……..

G20. How long does it take to put <twin1 name> and <twin2 name> to sleep in the evening? By this we mean how long it takes for the twins to fall asleep once they are in bed and ready to sleep.
Write hours and minutes. If less than one hour e.g. 15 minutes put 0 hours and 15 minutes. If 1 hour, put 1 hour and 0 minutes.
<twin1 name>: …….. hours …….. minutes
<twin2 name>: …….. hours …….. minutes

G21. When do <twin1 name> and <twin2 name> usually wake up in the morning?
Write hour : minutes using the 24 hour clock e.g. 6:15am should be 6:15.
<twin1 name>: …….. : ……..
<twin2 name>: …….. : ……..

G22. How long does <twin1 name> usually sleep during the daytime? Write hours and minutes. If less than one hour e.g. 15 minutes put 0 hours and 15 minutes. If 1 hour, put 1 hour and 0 minutes.

…….. hours …….. minutes per day

G23. How long does <twin2 name> usually sleep during the daytime? Write hours and minutes. If less than one hour e.g. 15 minutes put 0 hours and 15 minutes. If 1 hour, put 1 hour and 0 minutes.

…….. hours …….. minutes per day

G24. Does <twin1 name> usually wake up at night? If the participant says the child wakes up
sometimes or occasionally, check whether this is usually on a weekly basis. If they say yes, select yes.

Yes ☐ No ☐

G25. Does <twin2 name> usually wake up at night? If the participant says the child wakes up
sometimes or occasionally, check whether this is usually on a weekly basis. If they say yes, select yes.

Yes ☐ No ☐

If no for G24 and G25 skip to G28.and G29.

If yes for G24:

G26.1 How many nights in a normal week does <twin1 name> wake up?
0 1 2 3 4 5 6 7

G26.2 On the nights when <twin1 name> wakes up how many times does this happen?
……..

G26.3 How long per wake-up time does <twin1 name> stay awake at night?
Write hours and minutes. If less than one hour e.g. 15 minutes put 0 hours and 15 minutes. If 1 hour put 1 hour and 0 minutes.
…….. hours ……..minutes

If yes for G25:

G27.1 How many nights in a normal week does <twin2 name> wake up?
0 1 2 3 4 5 6 7

G27.2 On the nights when <twin2 name> wakes up how many times does this happen?
……..

G27.3 How long per wake-up time does <twin2 name> stay awake at night?
Write hours and minutes. If less than one hour e.g. 15 minutes put 0 hours and 15 minutes. If 1 hour put 1 hour and 0 minutes.
…….. hours ……..minutes

G28. Do you consider <twin1 name>'s sleep as a problem? The response options are 1 not at all, 2 a small problem, or 3 a serious problem.

Not at all ☐ A small problem ☐ or A serious problem ☐

G29. Do you consider <twin2 name>'s sleep as a problem? Again, the response options are 1 not at all, 2 a small problem, or 3 a serious problem (read this sentence aloud again if necessary).

Not at all ☐ A small problem ☐ or A serious problem ☐

Sections H - N: FOOD AVAILABLILTY

The next section is about food and drink that is currently in your home. For the food and drink that we ask about, please include all items that are in your home even if your twins don't eat or drink them. If you are unsure of any of the answers, please have a look to see what is in your home. If you have a phone in your kitchen and would like to move there now, that may help. Please answer as accurately as possible.

<u>Fruit</u>

H.1.1. Do you have any **fresh fruit** in your home now?

Yes ☐ No ☐

H.1.2. If yes, what types of **fresh fruit** do you have in your home now?
This is an open question. As the participant lists the fresh fruit they have, tick the matching options in the table or add any other fresh fruit to the free entry box which says other. When the participant finishes, prompt her/him by reminding her/him of places she/he may have forgotten: Have you remembered fresh fruit in your fridge, in a fruit bowl and in your cupboards?

Fresh fruit
List of standard fruits to choose from (see below) as well as a free-entry box for less common items.

	Yes / No
Apples	
Bananas	
Cherries	
Grapefruit	
Grapes	
Kiwi	
Mangoes	
Melon	
Nectarines	
Oranges	
Peaches	
Pears	
Pineapple	
Plums	
Strawberries	

Other fresh fruit	Number of other items

H.2.1. Do you have any **tinned or jarred fruit** in your home now?

Yes ☐ No ☐

H.2.2. If yes, what types of **tinned or jarred fruit** do you have in your home now?
This is an open question. As the participant lists the tinned or jarred fruit they have, tick the matching options in the table or add any other tinned or jarred fruit to the free entry box which says other.
When the participant finishes, prompt her/him by reminding her/him of places she/he may have forgotten: Have you remembered tinned or jarred fruit in your fridge and in your cupboards?

Tins / jars of fruit
List of standard fruits to choose from as well as a free-entry box for less common items.

	Yes / No
Cherries	
Fruit salad/cocktail	
Grapefruit	
Mandarin orange	
Peaches	
Pears	
Pineapple	
Plums	
Raspberries	
Strawberries	
Other	

Other tinned fruit	Number of other items

H.3.1. Do you have any **dried fruit**, such as raisins, dried apricots, or dates in your home now?

Yes ☐ No ☐

H.3.2. If yes, what types of **dried fruit** do you have in your home now?
This is an open question. As the participant lists the dried fruit they have, tick the matching options in the table or add any other dried fruit to the free entry box which says other. When the participant finishes, prompt her/him by reminding her/him of places she/he may have forgotten: Have you remembered dried fruit in a fruit bowl and in your cupboards?

Dried fruit
List of standard fruits to choose from as well as a free-entry box for less common items.

	Yes / No
Apples	
Apricots	
Banana chips	
Currants	
Dates	
Dried mixed fruit	
Prunes	
Raisins	
Sultanas	

Other dried fruit	Number of other items

H.4.1. Do you have any **frozen fruit** in your home now?

Yes ☐ No ☐

H.4.2. If yes, what types of **frozen fruit** do you have in your home now?
This is an open question. As the participant lists the frozen fruit they have, tick the matching options in the table or add any other frozen fruit to the free entry box which says other.

Frozen fruit
List of standard fruits to choose from as well as a free-entry box for less common items.

	Yes / No
Mixed berries	
Raspberries	
Strawberries	

Other frozen fruit	Number of other items

H.5.1. Would you say that the amount of **fruit** you currently have in your home is more than usual, less than usual, or about the same?

Less than usual ☐ The same ☐ More than usual ☐

H.5.2. Without opening any fridge or cupboard doors, is there **any kind of fruit** in your home now; displayed out in the open? A possible response may be that some fruit is behind a door, but it is a glass door and the fruit can be seen. If so, report YES. Another response could be that some fresh fruit is out, but that it is stored very high and can only be viewed with a stool. Is so, report NO.

Yes ☐ No ☐

H.5.3. Would it be possible for <twin1 name> and <twin2 name> to get any **fruit** by themselves, without your help? By this, we mean whether it would be physically possible for <twin1 name> and <twin2 name> to get any fruit by themselves, without your help.

If no, enter no for H5.4 and H5.5 and don't ask these two questions.

Yes ☐ No ☐

If different for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.

Is that for <twin1 name> or <twin2 name> or both?

H.5.4 Are <twin1 name> and <twin2 name> allowed to get any **fruit** by themselves, without your help or without asking you first?

Yes ☐ No ☐

If different for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.

Is that for <twin1 name> or <twin2 name> or both?

H.5.5 Do <twin1 name> and <twin2 name> ever get any fruit by themselves, without your help or without asking you first?

<twin1 name>: Yes ☐ No ☐

<twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.

Is that for <twin1 name> or <twin2 name> or both?

H.6. On average, how often do <twin1 name> and <twin2 name> eat fruit? This includes fruit that is eaten between meals and fruit that is eaten as part of a meal. Fruit juice is not included. This is an open question. Do not read the response options aloud but categorize the response accordingly. If the participant does not provide enough information e.g. they may say 'everyday', prompt for a fuller response e.g. 'so is that once a day, 2-3 times a day or 4 or more times a day?'

	Never or less than once a month	1-3 times a month	Once a week	2-4 times a week	5-6 times a week	Once a day	2-3 times a day	4 or more times a day
<twin1 name>	☐	☐	☐	☐	☐	☐	☐	☐
<twin2 name>	☐	☐	☐	☐	☐	☐	☐	☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Vegetables

K.1.1. Do you have any **fresh vegetables** in your home now? This includes salad items such as lettuce, cucumber, and tomato but not potatoes.

Yes ☐ No ☐

K.1.2. If yes, what types of **fresh vegetables** do you have in your home now?
This is an open question. As the participant lists the fresh vegetables they have, tick the matching options in the table or add any other fresh vegetables to the free entry box which says other.
When the participant finishes, prompt her/him by reminding her/him of places she/he may have forgotten: Have you remembered fresh vegetables in your fridge and in your cupboards?

Fresh vegetables
List of standard vegetables to choose from as well as a free-entry box for less common items.

	Yes / No
Broccoli	
Brussel sprouts	
Cabbage	
Carrots	
Cauliflower	
Celery	
Corn on the cob	
Cucumber	
Lettuce	
Mushrooms	
Onions	
Peppers	
Runner beans/green beans	
Swede	
Tomatoes	

Other fresh vegetables	Number of other items

K.2.1. Do you have any tinned or jarred vegetables for example tinned tomatoes, sweetcorn, or jarred beetroot, in your home now? This includes tinned pulses such as chickpeas, kidney beans and lentils.

Yes ☐ No ☐

K.2.2. If yes, what types of **tinned or jarred vegetables** do you have in your home now?
This is an open question. As the participant lists the tinned or jarred vegetables they have, tick the matching options in the table or add any other tinned or jarred vegetables to the free entry box which says other.
When the participant finishes, prompt her/him by reminding her/him of places she/he may have forgotten: Have you remembered tinned or jarred vegetables in your fridge and in your cupboards?

Tins of vegetables
List of standard vegetables to choose from as well as a free-entry box for less common items.

	Yes / No
Baked beans	
Bamboo shoots	
Beetroot	
Broad beans	
Carrots	
Mixed vegetables	
Mushrooms	
Peas	
Pease pudding	
Pickled onion	
Pickled gherkins	
Runner beans/green beans	
Sweetcorn	
Tomatoes	

Other tinned vegetables	Number of other items

K.3.1. Do you have any **frozen vegetables** in your home now?

Yes ☐ No ☐

K.3.2. If yes, what types of **frozen vegetables** do you have in your home now?
This is an open question. As the participant lists the frozen vegetables they have, tick the matching options in the table or add any other frozen vegetables to the free entry box which says other.

Frozen vegetables
List of standard vegetables to choose from as well as a free-entry box for less common items.

	Yes / No
Broad beans	
Brussel sprouts	
Cabbage	
Cauliflower	
Mange tout	
Mixed vegetables	
Peas	
Peppers	
Runner beans/green beans	
Spinach	
Sweet corn	

Other frozen vegetables	Number of other items

K.4.1. Would you say that the amount of **vegetables** you currently have in your home is more than usual, less than usual, or about the same?

Less than usual ☐ The same ☐ More than usual ☐

K.4.2. Do you have any ready to eat **fresh vegetables** on a shelf in the fridge or on the kitchen counter now? These include baby carrots, cherry tomatoes, or vegetables that you have sliced to make them ready to eat.

Yes ☐ No ☐

K.4.3. Would it be possible for <twin1 name> and <twin2 name> to get any **vegetables** by themselves without your help? By this, we mean whether it would be physically possible for <twin1 name> and <twin2 name> to get any vegetables by themselves, without your help.

If no, enter no for K4.4 and K4.5 and don't ask these two questions.

Yes ☐ No ☐

If different for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.

Is that for <twin1 name> or <twin2 name> or both?

K.4.4. Are <twin1 name> and <twin2 name> allowed to get any **vegetables** by themselves, without your help or without asking you first?

Yes ☐ No ☐

If different for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

K.4.5. Do <twin1 name> and <twin2 name> ever get any **vegetables** by themselves, without your help or without asking you first?

<twin1 name>: Yes ☐ No ☐

<twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

K.5. On average, how often do <twin1 name> and <twin2 name> eat vegetables? This includes salad items such as cucumber, lettuce and tomato but not potatoes. Vegetables that are eaten between meals and vegetables that are eaten as part of a meal are included.
This is an open question. Do not read the response options aloud but categorize the response accordingly. If the participant does not provide enough information e.g. they may say 'everyday', prompt for a fuller response e.g. 'so is that once a day, 2-3 times a day or 4 or more times a day?'

	Never or less than once a month	1-3 times a month	Once a week	2-4 times a week	5-6 times a week	Once a day	2-3 times a day	4 or more times a day
<twin1 name>	☐	☐	☐	☐	☐	☐	☐	☐
<twin2 name>	☐	☐	☐	☐	☐	☐	☐	☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.

Savoury snacks

L.1.1. Do you have any **savoury snacks** for example peanuts, crisps, tortillas and cheesy biscuits in your home now?

Yes ☐ No ☐

L.1.2. If yes, what types of **savoury snacks** do you have in your home now? Snacks like plain rice cakes, oatcakes, and breadsticks are not included.
This is an open question. As the participant lists the savoury snacks they have, tick the matching options in the table or add any other savoury snacks to the free entry box which says other.
When the participant finishes, prompt her/him by reminding her/him of places she/he may have forgotten: Have you remembered savoury snacks in your fridge and in your cupboards?

Savoury snacks

List of standard Savoury snacks to choose from as well as a free-entry box for less common items.

	Yes / No
Cheese biscuits	
Cheese straws	
Crisps	
Peanuts	
Pork scratchings	
Tortilla chips	

Other savoury snacks	Number of other items

L.1.3. Would you say that the amount of **savoury snacks** you currently have in your home is more than usual, less than usual, or about the same?

Less than usual ☐ The same ☐ More than usual ☐

L.2.1. Without opening any fridge or cupboard doors, are there any kind of **savoury snacks** in your home now; displayed out in the open? If yes, check that the participant is referring to snacks like crisps, peanuts and cheesy biscuits rather than snacks like plain rice cakes, oatcakes, and breadsticks. A possible response may be that some savoury snacks are behind a door, but it is a glass door and the snacks can be seen. If so, report YES. Another response could be that some savoury snacks are out, but that they are stored very high and can only be viewed with a stool. Is so, report NO.

Yes ☐ No ☐

L.2.2. Would it be possible for <twin1 name> and <twin2 name> to get any **savoury snacks** by themselves, without your help? By this, we mean whether it would be physically possible for <twin1 name> and <twin2 name> to get any savoury snacks by themselves, without your help. If yes, check that the participant is referring to snacks like crisps, peanuts and cheesy biscuits rather than snacks like plain rice cakes, oatcakes, and breadsticks. If no, enter no for L2.3 and L2.4 and don't ask these two questions.

Yes ☐ No ☐

If different for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

L.2.3. Are <twin1 name> and <twin2 name> allowed to get any **savoury snacks** by themselves, without your help or without asking you first? If yes, check that the participant is referring to snacks like crisps, peanuts and cheesy biscuits rather than snacks like plain rice cakes, oatcakes, and breadsticks.

Yes ☐ No ☐

If different for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

L.2.4. Do <twin1 name> and <twin2 name> ever get any **savoury snacks** by themselves, without your help or without asking you first? If yes, check that the participant is referring to snacks like crisps, peanuts and cheesy biscuits rather than snacks like plain rice cakes, oatcakes, and breadsticks.

<twin1 name>: Yes ☐ No ☐

<twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

L.2.5. On average, how often do <twin1 name> and <twin2 name> eat savoury snacks such as peanuts, crisps, tortillas and cheesy biscuits? This includes savoury snacks that are eaten between meals and savoury snacks that are eaten as part of a meal such as crisps with lunch. Again make sure that the participant is referring to snacks like crisps, peanuts and cheesy biscuits. This is an open question. Do not read the response options aloud but categorize the response accordingly. If the participant does not provide enough information e.g. they may say ‘everyday’, prompt for a fuller response e.g. ‘so is that once a day, 2-3 times a day or 4 or more times a day?’

	Never or less than once a month	1-3 times a month	Once a week	2-4 times a week	5-6 times a week	Once a day	2-3 times a day	4 or more times a day
<twin1 name>	☐	☐	☐	☐	☐	☐	☐	☐
<twin1 name>	☐	☐	☐	☐	☐	☐	☐	☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Any comments about savoury snacks

Sweet snacks

M.3.1. Do you have any **sweet snacks** for example cakes, biscuits or ice-cream in your home now?

Yes ☐ No ☐

M.3.2. If yes, what types of **sweet snacks** do you have in your home now? Do not include sweets or chocolate.
This is an open question. As the participant lists the sweet snacks they have, tick the matching options in the table or add any other sweet snacks to the free entry box which says other.
When the participant finishes, prompt her/him by reminding her/him of places she/he may have forgotten: Have you remembered sweet snacks in your fridge and in your cupboards?

Sweet snacks
List of standard sweet snacks to choose from as well as a free-entry box for less common items.

	Yes / No
Biscuits	
Buns	
Cakes	
Ice-cream	
Ice-lollies	
Pastries	

Other sweet snacks	Number of other items

M.3.3. Would you say that the amount of **sweet snacks** you currently have in your home is more than usual, less than usual, or about the same?

Less than usual ☐ The same ☐ More than usual ☐

M.4.1. Without opening any fridge or cupboard doors, are there any kind of **sweet snacks** in your home now displayed out in the open? If yes, check that the participant is referring to snacks like cakes, biscuits and ice cream. A possible response may be that some sweet snacks are behind a door, but it is a glass door and the snacks can be seen. If so, report YES. Another response could be that some sweet snacks are out, but that they are stored very high and can only be viewed with a stool. Is so, report NO.

Yes ☐ No ☐

M.4.2. Would it be possible for <twin1 name> and <twin2 name> to get any **sweet snacks** by themselves, without your help? By this, we mean whether it would be physically possible for <twin1 name> and <twin2 name> to get any sweet snacks by themselves, without your help. If yes, check that the participant is referring to snacks like cakes, biscuits and ice cream. If no, enter no for M4.3 and M4.4 and don't ask these two questions.

Yes ☐ No ☐

If different for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

M.4.3. Are <twin1 name> and <twin2 name> allowed to get any **sweet snacks** by themselves, without your help or without asking you first? If yes, check that the participant is referring to snacks like cakes, biscuits and ice cream.

Yes ☐ No ☐

If different for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

M.4.4. Do <twin1 name> and <twin2 name> ever get any **sweet snacks** by themselves, without your help or without asking you first? If yes, check that the participant is referring to snacks like cakes, biscuits and ice cream.

<twin1 name>: Yes ☐ No ☐

<twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

M.4.5. On average, how often do <twin1 name> and <twin2 name> eat sweet snacks such as cakes, biscuits, and ice-cream? This includes sweet snacks that are eaten between meals and sweet snacks that are eaten as part of a meal such as ice-cream for dessert. Again make sure that the participant is referring to snacks such as biscuits, cake, and ice cream. This is an open question. Do not read the response options aloud but categorize the response accordingly. If the participant does not provide enough information e.g. they may say 'everyday', prompt for a fuller response e.g. 'so is that once a day, 2-3 times a day or 4 or more times a day?'

	Never or less than once a month	1-3 times a month	Once a week	2-4 times a week	5-6 times a week	Once a day	2-3 times a day	4 or more times a day
<twin1 name>	☐	☐	☐	☐	☐	☐	☐	☐
<twin2 name>	☐	☐	☐	☐	☐	☐	☐	☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Any comments about sweet snacks.

Confectionery

N.1.1. Do you have any **confectionery** in your home now? This includes sweets and chocolate.

Yes ☐ No ☐

N.1.2. If yes, what types of **confectionery** do you have in your home now? This is an open question. As the participant lists the confectionery they have, tick the matching options in the table or add any other confectionery to the free entry box which says other.

When the participant finishes, prompt her/him by reminding her/him of places she/he may have forgotten: Have you remembered confectionery in your fridge, in a bowl and in your cupboards?

Confectionery

List of standard confectionery to choose from as well as a free-entry box for less common items.

	Yes / No
Chocolate	
Sweets	

Other confectionery	Number of other items

N.1.3. Would you say that the amount of **confectionery** you currently have in your home is more than usual, less than usual, or about the same?

Less than usual ☐ The same ☐ **2** More than usual ☐

N.2.1. Without opening any fridge or cupboard doors, is there any kind of **confectionery** in your home now displayed out in the open? A possible response may be that some confectionery is behind a door, but it is a glass door and the confectionery can be seen. If so, report YES. Another response could be that some confectionery is out, but that it is stored very high and can only be viewed with a stool. Is so, report NO.

Yes ☐ No ☐

N.2.2. Would it be possible for <twin1 name> and <twin2 name> to get any **confectionery** by themselves, without your help? By this, we mean whether it would be physically possible for <twin1 name> and <twin2 name> to get any confectionery by themselves, without your help.

If no, enter no for N2.3 and N2.4 and don't ask these two questions.

Yes ☐ No ☐

If different for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

N.2.3. Are <twin1 name> and <twin2 name> allowed to get any **confectionery** by themselves, without your help or without asking you first?

Yes ☐ No ☐

If different for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

N.2.4. Do <twin1 name> and <twin2 name> ever get any **confectionery** by themselves, without your help or without asking you first?

<twin1 name>: Yes ☐ No ☐

<twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

N.2.5. On average, how often do <twin1 name> and <twin2 name> eat confectionery such as chocolate and fruit sweets? This is an open question. Do not read the response options aloud but categorize the response accordingly. If the participant does not provide enough information e.g. they may say 'everyday', prompt for a fuller response e.g. 'so is that once a day, 2-3 times a day or 4 or more times a day?'

	Never or less than once a month	1-3 times a month	Once a week	2-4 times a week	5-6 times a week	Once a day	2-3 times a day	4 or more times a day
<twin1 name>	☐	☐	☐	☐	☐	☐	☐	☐
<twin2 name>	☐	☐	☐	☐	☐	☐	☐	☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Section O – FAST FOOD

O.1.1. On average, how often do <twin1 name> and <twin2 name> eat fast food from places such as McDonald's, KFC, Burger King, and Subway…? This includes both eating in and taking food away from fast food places. This is an open question. Do not read the response options aloud but categorize the response accordingly. If the participant does not provide enough information e.g. they may say 'everyday', prompt for a fuller response e.g. 'so is that once a day, 2-3 times a day or 4 or more times a day?'

	Never or less than once a month	1-3 times a month	Once a week	2-4 times a week	5-6 times a week	Once a day	2-3 times a day	4 or more times a day
<twin1 name>	☐	☐	☐	☐	☐	☐	☐	☐
<twin2 name>	☐	☐	☐	☐	☐	☐	☐	☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

O.1.2. On average, how often do <twin1 name> and <twin2 name> eat other convenience foods for their main meal? This includes food that requires no preparation such as ready-made pizza, microwaveable meals, and takeaway food such as fish and chips, Chinese, and Indian… Other convenience food such as fish fingers and chicken nuggets are included. This is an open question. Do not read the response options aloud but categorize the response accordingly. If the participant does not provide enough information e.g. they may say 'everyday', prompt for a fuller response e.g. 'so is that once a day, 2-3 times a day or 4 or more times a day?'

	Never or less than once a month	1-3 times a month	Once a week	2-4 times a week	5-6 times a week	Once a day	2-3 times a day	4 or more times a day
<twin1 name>	☐	☐	☐	☐	☐	☐	☐	☐
<twin2 name>	☐	☐	☐	☐	☐	☐	☐	☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Section P - DRINKS

P.1.1. Do you have any *non-alcoholic* **drinks** other than water in your home now?

If no to P1.1, skip P1.2. and P1.4. – P2.1. (but do ask P1.3. and P2.2.)

Yes ☐ No ☐

P.1.2. If yes, what types of non-alcoholic drinks do you have in your home now?
This is an open question. As the participant lists the drinks they have, tick the matching options in the table. May need to prompt to determine whether each drink is sugar sweetened or not.
When the participant finishes, prompt her/him by reminding her/him of places she/he may have forgotten: Have you remembered non-alcoholic drinks in your fridge and in your cupboards? Also need to prompt to make sure the participant has covered all non-alcoholic drink types e.g. if they don't mention milk say 'do you have any milk in your home now?'

	Sugar sweetened: (Yes / No)		Pure juice/No added sugar/diet: (Yes / No)
Fruit juice e.g. orange, apple			
Squash/cordial e.g. Robinson's blackcurrant cordial			
Fizzy pop e.g. coke, lemonade			
Ready made fruit flavoured drinks e.g. Ribena, Oasis			
Smoothies			
	Skimmed: (yes / no)	Semi-skimmed: (yes / no)	Full-fat: (yes / no)
Milk			

P.1.3. Would you say that the amount of non-alcoholic **drinks** you currently have in your home is more than usual, less than usual, or about the same?

Less than usual ☐ The same ☐ More than usual ☐

P.1.4. Without opening any fridge or cupboard doors, are there any non-alcoholic drinks in your home now; displayed out in the open? A possible response may be that some drinks are behind a door, but it is a glass door and the drinks can be seen. If so, report YES. Another response could be that some drinks are out, but that they are stored very high and can only be viewed with a stool. Is so, report NO.

Yes ☐ No ☐

P.1.5. If yes, what types of non-alcoholic drinks are displayed out in the open?
This is an open question. As the participant lists the drinks they have, tick the matching drink type in the table. May need to prompt the participant to determine whether each drink is sugar sweetened or not. For example, if the participant just says 'coke' interviewer says 'is that diet coke?' If the participant just says 'orange juice' interviewer says 'is that with added sugar?' etc.

	Sugar sweetened: (yes / no)		Pure juice/No added sugar/diet: (yes / no)
Fruit juice e.g. orange, apple			
Squash/cordial e.g. Robinson's blackcurrant cordial			
Fizzy pop e.g. coke, lemonade			
Ready made fruit flavoured drinks e.g. Ribena, Oasis			
Smoothies			
	Skimmed: (yes / no)	Semi-skimmed: (yes / no)	Full-fat: (yes / no)
Milk			

P.1.6. Would it be possible for <twin1 name> and <twin2 name> to get any non-alcoholic drinks by themselves, without your help? By this, we mean whether it would be physically possible for <twin1 name> and <twin2 name> to get any non-alcoholic drinks by themselves, without your help. If no, skip P1.7 - P2.1 and enter no for P1.8 and P2.

Yes ☐ No ☐

If different for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

P.1.7. If yes, what types of non-alcoholic drinks would be possible for <twin1 name> and <twin2 name> to get by themselves, without your help?

This is an open question. As the participant lists the drinks they have, tick the matching drink type in the table. May need to prompt the participant to determine whether each drink is sugar sweetened or not.

P.1.8. Are <twin1 name> and <twin2 name> allowed to get any non-alcoholic drinks by themselves, without your help or without asking you first?

Yes ☐ No ☐

If different for twins: <twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

P.1.9. If yes, what types of non-alcoholic drinks are <twin1 name> and <twin2 name> allowed to get by themselves, without your help or without asking you first?
This is an open question. As the participant lists the drinks they have, tick the matching drink type in the table. May need to prompt the participant to determine whether each drink is sugar sweetened or not.

P.2.0. Do <twin1 name> and <twin2 name> ever get any non-alcoholic drinks by themselves, without your help or without asking you first?

<twin1 name>: Yes ☐ No ☐

<twin2 name>: Yes ☐ No ☐

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Section Q - MEALTIMES

Note that for Q1.1 – Q5 the scores of <twin1 name> are automatically copied to <twin2 name>. If parent indicates a difference between twins, score can be adjusted for <twin2 name>. Always score <twin1 name> first and then <twin2 name>.

Q1.1. How many days a week do <twin1 name> and <twin2 name> eat **breakfast** at home? Weekly estimates include week days and weekend days. Breakfasts that are prepared at home, but not eaten at home do not count.

If 7 days, skip Q1.2. If the participant gives a number less than 7 e.g. 4, make sure they are always asked Q1.2 as it cannot be assumed that the twins eat breakfast on each of the remaining days elsewhere.

0 1 2 3 4 5 6 7 (days a week)

If different for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Q1.2. How many days a week do <twin1 name> and <twin2 name> eat **breakfast** elsewhere for example at nursery or preschool? This includes food prepared at home, foods purchased on the way to nursery or preschool and food prepared by the nursery or preschool – provided they are eaten outside the home.

0 1 2 3 4 5 6 7 (days a week)

If different for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Q1.3. How many days a week do your family sit at a table to eat **breakfast** together? This includes occasions when it is just <twin1 name> or <twin2 name> and yourself. Only include occasions where you or your <husband/wife/partner> actually eat with your twins. A possible response might be that they sit down as a family to eat breakfast, but not at a dining table. This is not included. Another possible response is that the twins sit at a table to eat breakfast with their nanny or other siblings, but not their parent(s). This is not included.

0 1 2 3 4 5 6 7 (days a week)

If different for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Q2.1. How many days a week do <twin1 name> and <twin2 name> eat a **midday meal** at home? Midday meals that are prepared at home, but not eaten at home do not count.

If 7 days, skip Q2.2. If the participant gives a number less than 7 e.g. 4, make sure they are always asked Q2.2 as it cannot be assumed that the twins eat a midday meal on each of the remaining days elsewhere.

0 1 2 3 4 5 6 7 (days a week)

If different for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.

Is that for <twin1 name> or <twin2 name> or both?

Q2.2. How many days a week do <twin1 name> and <twin2 name> eat a **midday meal** elsewhere for example at nursery or preschool? This includes food prepared at home, foods purchased on the way to nursery or preschool and food prepared by the nursery or preschool – provided they are eaten outside the home.

0 1 2 3 4 5 6 7 (days a week)

If different for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.

Is that for <twin1 name> or <twin2 name> or both?

Q2.3. How many days a week do your family sit at a table to eat a **midday meal** together? This includes occasions when it is just <twin1 name> or <twin2 name> and yourself. Only include occasions where you or your <husband/wife/partner> actually eat with your twins. A possible response might be that they sit down as a family to eat a midday meal, but not at a dining table. This is not included. Another possible response is that the twins sit at a table to eat a midday meal with their nanny or other siblings, but not their parent(s). This is not included.

0 1 2 3 4 5 6 7 (days a week)

If different for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.

Is that for <twin1 name> or <twin2 name> or both?

Q3.1. How many days a week do <twin1 name> and <twin2 name> eat an **evening meal** at home? Evening meals that are prepared at home, but not eaten at home do not count.

If 7 days, skip Q3.2. If the participant gives a number less than 7 e.g. 4, make sure they are always asked Q3.2 as it cannot be assumed that the twins eat an evening meal on each of the remaining days elsewhere.

0 1 2 3 4 5 6 7 (days a week)

If different for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Q3.2. How many days a week do <twin1 name> and <twin2 name> eat an **evening meal** elsewhere for example at nursery or preschool? This includes food prepared at home, foods purchased on the way to nursery or preschool and food prepared by the nursery or preschool – provided they are eaten outside the home.

0 1 2 3 4 5 6 7 (days a week)

If different for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Q3.3. How many days a week do your family sit at a table to eat an **evening meal** together? This includes occasions when it is just <twin1 name> or <twin2 name> and yourself. Only include occasions where you or your <husband/wife/partner> actually eat with your twins. A possible response might be that they sit down as a family to eat an evening meal, but not at a dining table. This is not included. Another possible response is that the twins sit at a table to eat an evening meal with their nanny or other siblings, but not their parent(s). This is not included.

0 1 2 3 4 5 6 7 (days a week)

If different for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Q4.1. How many days a week do <twin1 name> and <twin2 name> eat **snacks** at home? Snacks that are prepared at home, but not eaten at home do not count.

If 7 days, skip Q4.2. If the participant gives a number less than 7 e.g. 4, make sure they are always asked Q4.2 as it cannot be assumed that the twins eat snacks on each of the remaining days elsewhere.

0 1 2 3 4 5 6 7 (days a week)

If different for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.

Is that for <twin1 name> or <twin2 name> or both?

Q4.2. How many days a week do <twin1 name> and <twin2 name> eat **snacks** elsewhere for example at nursery or preschool? This includes snacks prepared at home, snacks purchased on the way to nursery or preschool and snacks prepared by the nursery or preschool – provided they are eaten outside the home.

0 1 2 3 4 5 6 7 (days a week)

If different for twins: <twin2 name>: 0 1 2 3 4 5 6 7

If the participant does not indicate whether their response is the same or different for <twin1 name> and <twin2 name>, prompt them to check.
Is that for <twin1 name> or <twin2 name> or both?

Section R – FOOD SHOPPING

R1.1. How often do you shop for food? This is an open question. Do not read the response options aloud but categorize the response accordingly. If the participant does not provide enough information the interviewer should prompt for a fuller response. For example, if the participant says ‘monthly big trip’, the interviewer should say ‘so is that with few small trips or no small trips?’ If participants say they do online shopping, also categorize their response according to the following options.

- ☐ Monthly, big trip, no small trips
- ☐ Monthly, big trip, few small trips
- ☐ Every other week, big trip, no small trips
- ☐ Every other week, big trip, few small trips
- ☐ Weekly, big trip, no small trips
- ☐ Weekly, big trip, few small trips
- ☐ As and when, no big trip, all small trips as needed
- ☐ Twice each week, big trips, no small trips
- ☐ Twice each week, big trips, few small trips

R1.2. How often do <twin1 name> and <twin2 name> go food shopping with you? Please choose a score from 1 to 5: 1 means never, 2 means rarely, 3 means some of the time, 4 means most of the time, 5 means all of the time? Participants may respond before you get a chance to read them the options. Let them finish and then say, ‘ok, can you tell me whether this happens 1 never, 2 rarely…etc.’

1 2 3 4 5 (5=all of the time)

If different arrangement for twins: <twin2 name>: 1 2 3 4 5

R1.3. How many days has it been since you last shopped for food? If participants only do online shopping, which is then delivered, make sure they are asked about how many days it has been since food was last *delivered* to their house. Participants may say that they have done their next online shop recently but we want to know how many days it has been since food shopping came into the house.

…… days

R1.4. Was the last shop small or big?

Small ☐ Big ☐ Medium ☐

Section S – ADDITIONAL GENERAL INFORMATION QUESTIONS

Finally, the last few questions are about your twin's growth.

Height and Weight

S1. Do you have any recent height or weight measurements for <twin1 name> or <twin2 name>? The most recent measurements we have were taken on (date automatically inserted).

Yes ☐ No ☐

Parents may have already taken measurements recently but have not yet sent them in. If so, just add the measurements to the tables. Otherwise, ask as below.

S4. Would now be a convenient time to take the twins' height and weight measurements?

If yes, Using the height chart and scales we have sent, please can you take today's heights and weights for each of the twins and then read them out to me? Please remember to measure and weigh the twins in indoor clothes without shoes.

Once the twins have been measured and weighed add the measurements to the table at the bottom of the page.

If no, Would you be able to take these measurements tomorrow?

If yes, Please use the height chart and scales we have sent to take the twins heights and weights. Remember to measure and weigh the twins in indoor clothes without shoes. Once you have taken these measurements, please send them to us by email, give them over the telephone, or add them on the Gemini website.

How would you like to give these measurements?

If email, please email to Gemini@ucl.ac.uk
(make sure participant includes their Gemini ID number and the date the measurements were taken).

If telephone, please call 020 7679 1723.

If Gemini website, please go to www.attitudestohealth.co.uk/gemini and click where it says enter height/weight measurements.

If no, when would be a convenient time for you to take these measurements? Repeat the text beneath tomorrow's measurements, making sure you record how participants will give the measurements and when they will give them.

That's the end of the interview now. Thank you very much for your time. Do you have any questions or comments?

Appendix E CHAOS

YOUR HOME ENVIRONMENT
Please read the following statements and indicate whether each statement is true or false for your home

		True	False
D1.	We almost always seem to be rushed	☐	☐
D2.	It's a real zoo in our home	☐	☐
D3.	There is often a fuss going on at our home	☐	☐
D4.	You can't hear yourself think in our home	☐	☐
D5.	Our home is a good place to relax	☐	☐
D6.	The atmosphere in our home is calm	☐	☐

D7. **The Gemini team is currently applying for funding for a health assessment that would be carried out at a clinical research centre in London. If we are successful in obtaining funding for this research, do you think you would be interested in this part of the study?**

Yes ☐ No ☐ Unsure ☐

Appendix F Eating behaviour: CEBQ

11.1 CEBQ

SR

How would you describe your twins' eating styles on a typical day?

			Never	Rarely	Sometimes	Often	Always
C9.	My child loves food	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
C10.	My child eats more when worried	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
C11.	My child wants to eat (e.g. asks) when he/she <u>sees</u> certain foods	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
C12.	My child has a big appetite	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
C13.	My child finishes his/her meal quickly	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐

			Never	Rarely	Sometimes	Often	Always
C14.	My child is interested in food	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
C15.	My child is always asking for a drink	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
C16.	My child refuses new foods at first	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
C17.	My child eats slowly	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
C18.	My child eats less when angry	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
C19.	My child enjoys tasting new foods	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
C20.	My child wants to eat (e.g. asks) when he/she smells certain foods	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
C21.	My child eats less when he/she is tired	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
FR C22.	My child is always asking for food	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
C23.	My child eats more when annoyed	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
FR C24.	If allowed to, my child would eat too much	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐

				Never	Rarely	Sometimes	Often	Always
	C25.	My child eats more when anxious	1st born	☐	☐	☐	☐	☐
			2nd born	☐	☐	☐	☐	☐
	C26.	My child enjoys a wide variety of foods	1st born	☐	☐	☐	☐	☐
			2nd born	☐	☐	☐	☐	☐
SR	C27.	My child leaves food on his/her plate at the end of a meal	1st born	☐	☐	☐	☐	☐
			2nd born	☐	☐	☐	☐	☐
	C28.	My child takes more than 30 minutes to finish a meal	1st born	☐	☐	☐	☐	☐
			2nd born	☐	☐	☐	☐	☐
FR	C29.	Given the choice, my child would eat most of the time	1st born	☐	☐	☐	☐	☐
			2nd born	☐	☐	☐	☐	☐
	C30.	My child looks forward to mealtimes	1st born	☐	☐	☐	☐	☐
			2nd born	☐	☐	☐	☐	☐
SR	C31.	My child gets full before his/her meal is finished	1st born	☐	☐	☐	☐	☐
			2nd born	☐	☐	☐	☐	☐
	C32.	My child enjoys eating	1st born	☐	☐	☐	☐	☐
			2nd born	☐	☐	☐	☐	☐
	C33.	My child eats more than usual if he/she really enjoys the taste of a food	1st born	☐	☐	☐	☐	☐
			2nd born	☐	☐	☐	☐	☐
	C34.	My child eats more when he/she is happy	1st born	☐	☐	☐	☐	☐
			2nd born	☐	☐	☐	☐	☐
	C35.	My child is difficult to please with meals	1st born	☐	☐	☐	☐	☐
			2nd born	☐	☐	☐	☐	☐

			Never	Rarely	Sometimes	Often	Always
	C36. My child eats less when upset	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
SR	C37. My child gets full up easily	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
	C38. My child eats more when he/she has nothing else to do	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
FR	C39. Even if my child is full up he/she finds room to eat his/her favourite food	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
	C40. If given the chance, my child would drink continuously throughout the day	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
SR	C41. My child cannot eat a meal if he/she has had a snack just before	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
	C42. If given the chance, my child would always be having a drink	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
	C43. My child is interested in tasting food he/she hasn't tasted before	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
	C44. My child wants to eat (e.g. reaches out or asks for food) when he/she sees others eating	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐
	C45. My child decides that he/she doesn't like a food without even tasting it	1st born	☐	☐	☐	☐	☐
		2nd born	☐	☐	☐	☐	☐

FR

		Never	Rarely	Sometimes	Often	Always
C46. If given the chance my child would always have food in his/her mouth	1st born	☐	☐	☐	☐	☐
	2nd born	☐	☐	☐	☐	☐
C47. My child eats more and more slowly during the course of a meal	1st born	☐	☐	☐	☐	☐
	2nd born	☐	☐	☐	☐	☐

Appendix G Study 1: additional tables

12.1 Table I.1. Logistic regression models predicting shorter nighttime sleep, when considered as sleep <10th percentile

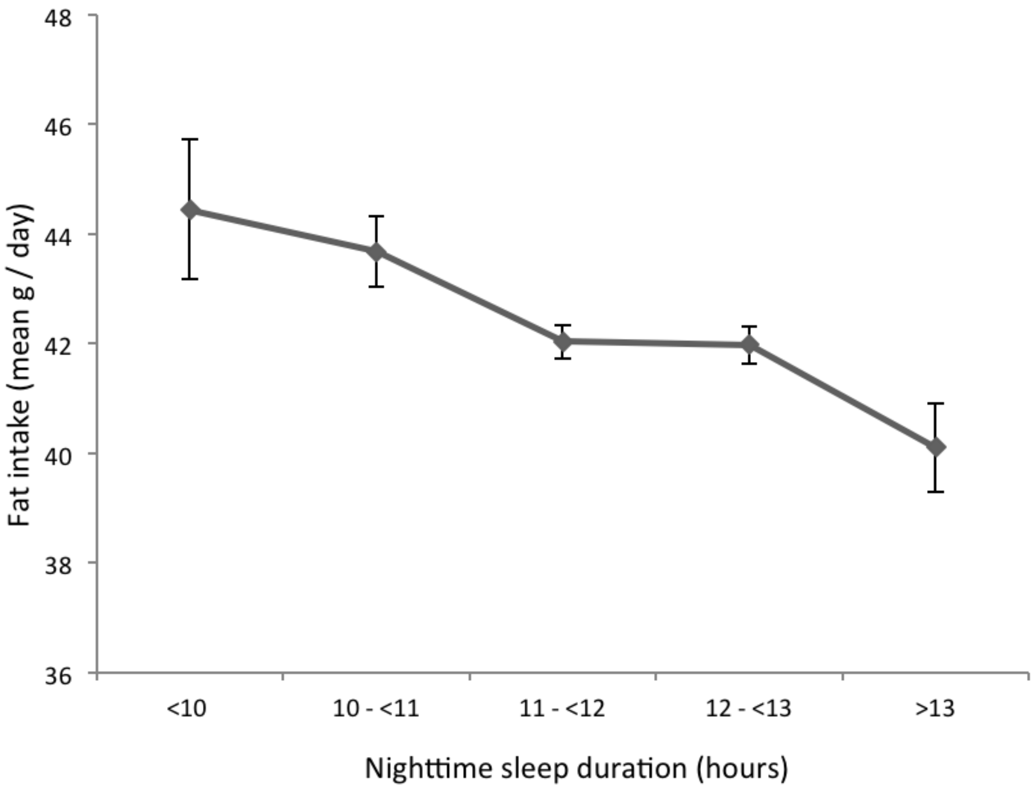
Table I.1. Logistic regression models predicting shorter night time sleep, when considered as sleep <10th percentile

Risk Factors	OR (95% CI)
Total (n=1702)	
Maternal education	
High (n=964)	1.00
Low (n=738)	1.61 (1.16 to 2.25)*
Ethnicity	
White (n=1509)	1.00
Non-White (n=193)	3.90 (2.66 to 5.72)**
Sex	
Female (n=874)	1.00
Male (n=828)	1.37 (1.00 to 1.88)*
Birth weight (grams)	
> 2500 (n=858)	1.00
≤ 2500 (n=844)	1.30 (0.94 to 1.79)
Older children	
0 (n=889)	1.00
1 (n=563)	0.95 (0.66 to 1.38)
>1 (n=250)	2.01 (1.33 to 3.04)*
Morning TV (hours)	
≤1 (n=1176)	1.00
>1 (n=526)	1.31 (0.92 to 1.87)
Evening TV (hours)	
≤1 (n=1489)	1.00
>1 (n=213)	2.08 (1.37 to 3.17)*
Mediators	
Wake time (per hour)	0.17 (0.13 to 0.23)**
Bedtime (per hour)	6.54 (4.96 to 8.62)**

Abbreviations: *p<0.05; **p≤0.001; OR, odds ratio; CI, confidence interval

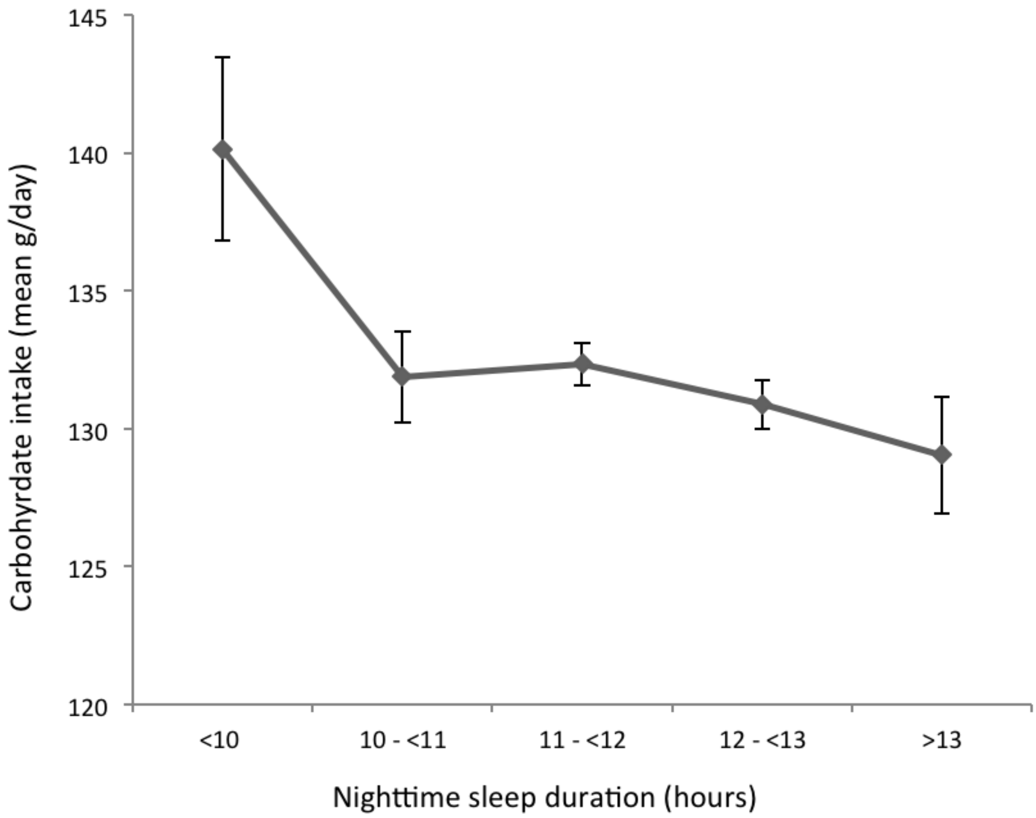
Appendix H Study 2: additional tables and figures

13.1 Figure J.1. Adjusted fat intake by nighttime sleep duration



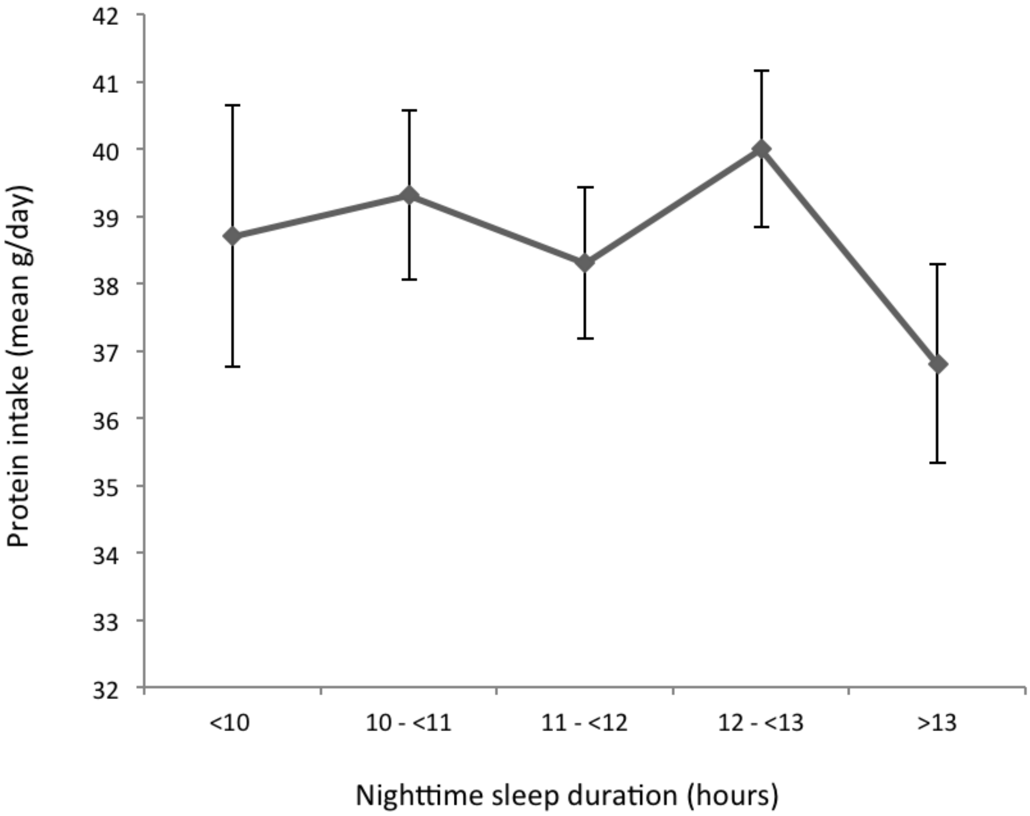
P for linear trend =0.013

14.1 Figure J.2. Adjusted carbohydrate intake by nighttime sleep duration



P for linear trend = 0.006

15.1 Figure J.3. Adjusted protein intake by nighttime sleep duration



P for linear trend = 0.306

16.1 Table J.1 Weight adjusted dietary data by nighttime sleep duration

Table J.1. Weight adjusted dietary data by nighttime sleep duration*. Data given as mean (standard error)

Hours	**<10**	**10 - <11**	**11 - <12**	**12 - <13**	**>13**	**P (linear trend)**
Number (total n=1303)	25	116	501	384	68	
Total energy intake (kcal/day)	1050.9 (45)	1010.3 (29)	981.4 (26)	978.7 (27)	931.4 (34)	0.003

*Data adjusted for weight SDS measured between 15-24 months of age. Model also fully adjusted for all covariates (age, sex, maternal education, and daytime sleep)

Appendix I Study 3: additional tables

17.1 Table K.1. Adjusted dietary data by nighttime sleep duration

Table K.1. Adjusted dietary data by nighttime sleep duration (mean (standard error)). Models adjusted for age, sex, maternal education, birth weight and daytime sleep

	< 10 hours	10 - < 11 hours	11 - < 12 hours	12 - < 13 hours	≥ 13 hours	p (linear trend)
Total energy intake by eating occasion (mean kcal per day)						
Meals	710.4 (33.0)	672.1 (16.9)	707.4 (7.9)	706.8 (9.4)	702.1 (21.3)	0.823
Snacks	170.1 (18.8)	163.7 (9.6)	152.5 (4.6)	154.7 (5.4)	142.1 (12.2)	0.161
Drinks only	175.9 (21.5)	178.1 (11.0)	142.3 (5.2)	149.4 (6.1)	135.0 (13.9)	0.036
Total energy intake by time of day (mean kcal per day)						
Morning	266.9 (16.2)	269.6 (8.3)	281.0 (3.9)	279.0 (4.6)	259.8 (10.5)	0.903
Day	350.1 (17.4)	328.3 (8.9)	325.3 (4.2)	335.7 (4.9)	341.5 (11.2)	0.820
Afternoon/Evening	318.4 (20.3)	326.9 (10.4)	346.9 (4.9)	357.7 (5.8)	359.2 (13.1)	0.024
Night	164.9 (14.0)	113.3 (7.2)	76.8 (3.4)	59.6 (4.0)	45.9 (9.1)	<0.001
Total energy intake at night by eating occasion (mean kcal per night)						
Meals	25.7 (6.0)	10.9 (3.1)	7.6 (1.5)	4.4 (1.7)	5.0 (3.9)	0.001
Snacks	22.4 (4.1)	11.4 (2.1)	3.7 (1.0)	4.0 (1.2)	0.9 (2.7)	<0.001
Drinks only	116.9 (12.1)	91.0 (6.2)	65.4 (2.9)	51.1 (3.4)	40.0 (7.8)	<0.001

Appendix J Study 4: additional tables

18.1 Table L.1 Comparison between groups with and without BMI data for each key variable used in Study 4

Table L.1. Mean (standard deviation) for each key variable by sample. P-value for between group differences.

	Sample without BMI data at age 5 years	Sample with BMI data age 5 years	P value for between group difference
N	514	494	
Birth weight (kg)	2.46 (0.54)	2.46 (0.54)	0.99
Food Responsiveness	2.42 (0.76)	2.34 (0.73)	0.10
Satiety Responsiveness	2.83 (0.63)	2.85 (0.60)	0.76
Nighttime sleep	11.47 (0.71)	11.48 (0.60)	0.86

Appendix K Publications relating to work in this thesis

19.1 Publication of Study 1

[redacted]

Appendix L Courses and conferences attended

Conferences I have presented at during my PhD

The Obesity Society (TOS), Annual Congress, November 2014, Boston USA.
McDonald L, Wardle J, Llewellyn CH, Fisher A. The role of food responsiveness in moderating the association between sleep duration and BMI in early life (poster presention).

Association for the study of Obesity (ASO), Annual National Congress, September 2014, University of Birmingham**.**
McDonald L, Wardle J, Llewllyn CH, Johnson L, van Jaarsveld CHM, Fisher A. Sleep and energy intake at night in early childhood (poster presentation).

20th European Congress on Obesity (ECO2013), May 2013, Liverpool.
McDonald L, Wardle J, Llewllyn CH, van Jaarsveld CHM, Fisher A. Shorter sleep is associated with higher energy intake in infants (poster presentation).

Other conferences I have attended and courses completed during my PhD

HBRC conference, October 2014, UCL.
Day conference that provided an opportunity to share the research that has been done by the three research groups within the HBRC and to learn more about the skills and expertise of the members of various groups.

MRC Social, Genetic and Developmental Psychiatry (SGDP) Twin Model-Fitting Course (Open Mx), June 2013, at the SGDP Summer School, Institute of Psychiatry, Kings College London.

Social, Biological and Environmental Exposures and Health across the Life Course, June 2013, at Cumberland Lodge, Windsor.
Three day conference for PhD students in the department of Epidemiology and Public Health at UCL. Speakers included Professor Jane Wardle, Professor Russell Viner, Professor Yvonne Kelly, and Professor Andrew Steptoe.

'Perils and Promises of Dieting', November 2012, UCL.
A half-day event hosted by the charity Weight Concern on behalf of TEMPEST (Temptations to Eat Moderated by Personal and Environmental Self-regulatory Tools' a European Commission-funded project on healthy eating.) Conference focused on the current obesity epidemic, the ethical debate surrounding obesity, and resisting temptations to eat in the current food environment. This event was chaired by Vivienne Parry OBE.